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EPIDEMIC ENCEPHALITIS.[1]

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DURING recent years epidemic encephalitis has been a fertile source of contribution to medical literature abroad, but very little has appeared in the Australian journals. The subject for tonight's discussion should therefore prove of unusual interest. The disease has been well known in Sydney in its well established state, but its early recognition is at times fraught with considerable difficulty. This is owing to its occasional insidious onset, with symptoms resembling those of influenza. In the short time at my disposal only certain features of this widely variable disease can be touched upon. I propose to confine my remarks to the following considerations: ætiology, clinical features, sequelæ and treatment.

Ætiology.

A large amount of experimental work has been done and the results published. The work is all similar and falls under two headings: (i) Levaditi, Doerr, Loewy and Straus, Kling and others have injected into monkeys and rabbits pulped brain and naso-pharyngeal discharges of human beings who had suffered from epidemic encephalitis. They have in a proportion of cases reproduced encephalitis in the injected animals. It has been possible to pass the disease from the first into a second series of animals.

(ii) Szymanowski and Zylberblast-Zand[1] and others have endeavoured to prove the identity of the virus of epidemic encephalitis with that of *herpes febrilis*. They used rabbits for their experiments. All the rabbits inoculated with herpetic virus developed encephalitis, while the disease appeared in 33% of the rabbits injected with the virus of *encephalitis lethargica*.

The pathological condition of the brains found *post mortem* was identical in the two series of experiments.

Greenfield[2] gives an excellent review of the experimental work published upon this aspect of the subject. He states that when rabbits were used as the animals for experiment 50% yielded positive results and when attempts were made to re transmit the disease to other animals, again 50% of positive results were obtained. It seems agreed then that 50% of animals inoculated with human virus develope encephalitis. But he throws doubt upon the interpretation of these experiments by pointing out that laboratory rabbits are susceptible to a spontaneous form of encephalitis due to a protozoan, named by Levaditi *Encephalitozoon cuniculi*. Greenfield reports that McCartney examined 372 rabbits in the Rockefeller Institute and found 55% suffering from encephalitis due to *Encephalitozoon cuniculi*. No finality, therefore, can be considered established concerning the ætiology of epidemic encephalitis.

From the published work it appears that the virus of this disease is an ultra-microscopic, filter-passing organism. There is evidence from many sources that it has been possible to communicate the disease to laboratory animals and through them to other animals. The virus is neurotropic, that is from any portal of entry to the body it ultimately establishes itself and produces its pathological effects within the nervous system. In this respect it resembles the tetanus bacillus. Further there is evidence that the virus of epidemic encephalitis is identical with that of *herpes febrilis*.

Clinical Features.

One outstanding fact must be emphasized and that is that the disease may manifest itself in widely varying ways. Epidemic encephalitis is always a serious threat to life. There appears to be a fatal issue in about one-quarter of all cases. The incubation period is probably short and has been stated by Hall as from one to fourteen days. Boyd[3] quotes a case in which there definitely appears to have been an incubation of fourteen days. There are few general or constitutional symptoms and these appear early. They consist of fever, furred tongue and general malaise. The important signs and symptoms may be regarded not as the result of toxæmia, but as directly due to the pathological lesion produced in the central nervous system. Usually these are few, sometimes they are so slight as to pass unrecognized and are recalled only by an effort of memory when at a later date treatment is sought for recognizable sequelæ.

Lethargy is common. It is generally an early symptom, but may not appear for months. The patient appears to be sleeping quietly, but can be roused and fed by mouth. The lethargy may last for weeks or months. The patient may recover from this lethargy only to develope it again. In some epidemics, however, as in the second Winnipeg outbreak recorded by Boyd, lethargy is not a symptom, but instead great restlessness is seen.[3]

Paralysis of all kinds may occur. In Sydney we have come to regard transient diplopia due to temporary palsies of the eye muscles as typical of this disease. The palsies may cause ptosis, a dilated pupil or strabismus. There may be facial or hypoglossal paralysis. There may be hemiplegia or even paraplegia, both of which, however, are extremely rare. Bradykinesia is common along with such involuntary movements as choreiform movements, myoclonic movements and tremor. Respiratory disorders occur and include tachypnœa, hiccup, yawning *et cetera*.

I have analysed the case records of fifty patients admitted consecutively to the Royal Prince Alfred Hospital, Sydney, during the years 1919 to 1927. Nine of these patients died in hospital. All but nine were lethargic in hospital. Of these nine one was lethargic prior to admission. A Babinski reflex

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and the Section of Neurology and Psychiatry of the Branch on May 31, 1928.

was elicited in only four patients. Irregular movements were recorded in the histories of ten patients and convulsions in those of three. The vast majority gave evidence of cranial nerve involvement. Mental symptoms were rare. The cerebro-spinal fluid of every patient examined was sterile and no abnormality beyond increase in quantity was determined. In one patient, however, it is recorded that there was definitely an increase of lymphocytes.

The cytology of the blood was not fully inquired into, but in some patients there had been a slight neutrophile leucocytosis, in others no alteration. The largest count was one of 18,000 leucocytes, of which 84% were neutrophile, in a patient who had been ill only one week. The blood of one patient only yielded a Wassermann reaction. In no case examined did the cerebro-spinal fluid yield a Wassermann reaction.

The lesions in epidemic encephalitis are found chiefly in the mid-brain and pons and the basal nuclei and produce their most lasting effect in that part of the brain known as the extrapyramidal motor system. The blood vessels of the affected portion of the brain are enlarged and surrounded by an area of cellular infiltration. Thromboses and hæmorrhages occur. Whilst these vascular changes are found principally in the base of the brain, in many *post mortem* examinations similar changes have been found in the cerebrum. Calcification was found by Boyd and McAlpine in the walls and lumen of the vessels and in the brain substance of the *corpus striatum*. Degenerative changes may occur in affected nuclei. The pathological changes found in the extra-pyramidal motor system will be discussed later.

The Sequelæ.

The sequelæ consist of muscular paralysis and wasting, involuntary movements, respiratory disorders, endocrine disorders, general asthenia, the Parkinsonian syndrome and mental alienation. The chief endocrine disturbance seen is obesity, as in the case of one young woman treated at the Royal Prince Alfred Hospital who suffered also from Parkinsonian syndrome. Of all these the Parkinsonian syndrome is the only sequela which time will permit me to discuss. The facial immobility, muscular rigidity and occasional tremor associated with this condition are so well known as to require no detailed description. It may be rapid or slow in reaching full development. It may never progress to the extent of embarrassing the patient.

In many patients it appears during the initial illness as in some referred to in my analysis of the Royal Prince Alfred Hospital records. In others it may not appear till after a few years have elapsed. Occasionally a young patient is seen suffering from Parkinsonism who has no recollection of any parental infection.

In advanced cases the patient's chief disability is due to the extreme muscular rigidity which prevents him from performing delicate movements, such as dressing and undressing, writing and shaving. The rigidity affects all the muscles of the limbs and face. It may commence unilaterally. An illustrative case is A.C.J.S., aged forty years, who stated that in 1916 or 1917 he had an acute short illness with transient diplopia. In 1918 he developed rigidity and tremor of the right upper limb. By 1920 the right lower limb was affected and the Parkinsonian mask was obvious. Under treatment by belladonna he improved, only to relapse at a later date. Not until 1924 did the opposite upper and lower limbs commence to manifest signs of rigidity and tremor. Today his condition is a typical example of extreme Parkinsonism. A study of the underlying pathology of the patient has led to important contributions to our knowledge of the extrapyramidal motor system. Douglas McAlpine[4] has analysed an account of published cases and publishes the pathological findings in eight cases of Parkinsonism. He attributes the characteristic features of the syndrome to changes in the *substantia nigra* in which he found gliosis, cellular degeneration and destruction.

At this stage a consideration of the extrapyramidal motor system will be useful. This system consists of the *corpus striatum*, the red nucleus, the *nucleus hypothalami* of Luys and the *substantia nigra*. The connexions are uncertain. Heiller states that from the large efferent cell of the *globus pallidus* axones pass to the red nucleus, the nucleus of Luys, the *substantia nigra*, the thalamus and perhaps other tegmental nuclei.[5] From the red nucleus passes the rubro-spinal tract to influence the lower motor neurone. He thinks that probably other descending tracts similar in function proceed downwards from the nuclei of Luys and the *substantia nigra*. Kinneir Wilson regards the *corpus striatum* as controlling muscular tone and neuromuscular rhythm and interruption of its influence as leading to hypertonicity and tremors. It is possible, therefore, that the hypertonicity and bradykinesia of the Parkinsonian syndrome are of the nature of release phenomena. The question of their exact neurological basis, however, cannot be considered as finalized.

It is important to note that McAlpine found subacute inflammatory changes in the mid-brain in cases of long-established Parkinsonism. This raises the question of the duration of the parent infection. It is becoming commonly believed that the sequelæ of epidemic encephalitis would be better described as late manifestations of the disease. In other words epidemic encephalitis should not be likened to poliomyelitis and polyencephalitis which in an acute illness destroys certain parts of the central nervous system and dies out. Encephalitis also commences acutely and its severity is soon checked, but it subsequently smoulders in a chronic but active form for an undetermined length of time. Much evidence has been published in support of this view. We have the clinical evidence of the protracted course of the primary disease in some patients with relapses of lethargy and fever. Further the sequelæ may be very severe after even

the mildest form of primary infection. All authorities refer to the irregular course of the disease. Again the occasional very late appearance of Parkinsonism probably favours the belief in a latent infection rather than in progressive gliosis as a cause. Finally, Parkinsonism is, as is well known, by no means always progressive. It may progress to a certain point only. I am at present treating a patient at the Royal Prince Alfred Hospital for this syndrome, who is definitely better now than he was two years ago. Hall states that there is a tendency in some patients to improve slightly.[6] This consideration of the duration of the disease is very important from the point of view of prognosis and treatment.

Prognosis and Treatment.

The treatment of epidemic encephalitis during its early stages is unfortunately unsatisfactory. We know no treatment that will abort the disease or prevent the onset of sequelæ.

Whichever way we regard it, epidemic encephalitis must be regarded as a very serious disease. A. Gardner Robb analysed the fate of 173 patients who suffered from the 1924 epidemic of encephalitis in Belfast. Of these twenty-one died. Of the 152 who recovered, eleven were untraced. Of the remainder only thirteen were found to be well after two and a half years.[7] Geoffry Eden and A. Lowndes Yates[8] claimed good results both in acute cases and in late manifestation of the disease from washing out the nasal accessory sinuses; the infection is believed to enter the body at this situation and persistent infection can be maintained there. Lumbar puncture is not indicated unless cerebrospinal irritation is pronounced.

Success has been claimed for intravenous injections of sodium salicylate. Probably no treatment is successful. As far as the late manifestations are concerned, all that one can hope to do is to arrest this infection. It is obvious that no treatment can replace the cells that have disappeared from the *substantia nigra*. Both belladonna and hyoscine are used to minimize the hypertonicity of Parkinsonism. In my opinion hyoscine hydrobromide is preferable. I have seen one patient upon whom was performed a unilateral ramisection. The result was a slight but definite improvement in the limbs upon the side operated upon. I have had no experience of treatment by hexamine which is recommended to be given in large doses in association with alkalis, the latter as a precaution to protect the kidneys from damage. It is probable that in the near future more time will be devoted to research upon treatment than upon the underlying pathology.

In conclusion I wish to thank the record room staff and the honorary physicians of the Royal Prince Alfred Hospital for their kindness in granting me access to their medical histories.

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THE MENTAL ASPECTS OF ENCEPHALITIS.[1]

By W. S. DAWSON, M.D.,

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IN acute encephalitis we have to deal with mental symptoms which are in no way specific, but which are seen in a variety of conditions where the functions of the cerebrum are impaired through toxic or mechanical causes (trauma, tumour and vascular lesions). Bearing in mind the Jacksonian conception of the dissolution of functions, the mental symptoms, like those of a lower order in the functional hierarchy, namely the physical signs, may be considered as inhibition and release phenomena from the temporary or permanent suspension of activity on the part of the highest level. The outstanding mental feature in acute encephalitis, at any rate in hospital patients, is lethargy or stupor. The following case which came under my notice early in 1920, is typical.

Mental Symptoms in the Acute Stage.

CASE I.—F., aged twenty-two years, had complained of headaches for three months and felt very depressed for about two weeks before admission to a hospital in London. Quite suddenly her headaches increased in severity and her vision became blurred and she saw double. She also complained of some difficulty in swallowing and of inability to control the movements of her hands. She stated that her memory was hazy and that she saw objects turn into animals. On admission the temperature was 37·2° C. (99° F.), her respirations twenty-four and her pulse rate seventy-six. She was drowsy, could be roused with difficulty, muttered in her sleep and resented interference. She said that the furniture turned into animals and men. The physical signs included ptosis, paralysis of external ocular muscles, nystagmus in all directions and muscular spasms and transient plantar extensor responses. She remained in a dreamy lethargy with a placid expression for about a month after which she gradually improved and on discharge seemed perfectly well. Two years later she presented the Parkinsonian syndrome in a marked degreee.

A very similar mental symptomatology occurs in trypanosomiasis where quite early in the disease owing to increasing hebetude and drowsiness the

[1] Read at a meeting of the New South Wales Branch of the British Medical Association in conjunction with the Section of Medicine and the Section of Neurology and Psychiatry on May 31, 1928.

patient has to be roused and to be kept awake to take his meals.

The lethargy may be associated with overactivity in the form of delirium and restlessness. The delirium may assume an "occupational" type. For example, a child, aged seven years, with inversion of the sleep rhythm spent the whole night rearranging his bedding and when spoken to appeared dazed and only very dimly conscious of his surroundings. Another patient with a "wild" delirium continually gave telephone calls. Mapother[1] cites the case of a barman who repeatedly called out "Time, please, gentlemen." Sometimes the excitement may be extreme, amounting to acute delirious mania. These symptoms are all well known in alcoholic and other toxic states and are not peculiar to epidemic encephalitis. One or two patients have come under my notice displaying extreme terror.

CASE II.—A female, aged thirty-four years, had had a "fit" nine months previously; she was shaky for a few days and staggered about. For five months she had had difficulty in concentrating and her memory seemed impaired. She entered a general hospital, but became so disturbed that she was certified. Her certificate contained the following: "She is agitated, talks to herself, does not know where she is, her memory is grossly impaired, she appears to be in a state of fear and is full of delusions." When I saw her on admission to a mental hospital in England she lay in bed, her eyes staring and rolling in all directions; the mouth was held wide open; there was a lateral tremor of her head and neck; her arms were held stiffly to her sides, semiflexed, with tremors in her hands. Every few minutes she had a kind of seizure; a look of great distress came over her face and she cried out in terror. She then gave a gasp, the fists were clenched and the arms tightly flexed for a few seconds. After these tonic fits she said that she suddenly felt afraid of something, she did not know what, but sometimes she had a fearful vision just before or simultaneously with the spasms. She presented a number of physical signs, pyrexia and increase of small cells in the cerebro-spinal fluid. From excitement she passed into stupor from which she could be roused for short intervals, when she frequently complained of "goneness." She was discharged mentally clear, but with mild Parkinsonism.

This case illustrates the varied symptomatology which may occur during the course of the disease.

Some patients display a peculiar loquacity, pouring out an unending stream of talk, mostly incoherent, and lacking the emotional display and the appreciation of the relation to environment which characterize the utterances of those suffering from true mania. Profound depression with suicidal impulses has been noted in a few patients in the acute stage.

Since the mental symptoms of the acute stage for the most part in themselves lack any distinguishing features, one must depend upon the physical examination for an understanding of the case. Stupor may cause some difficulty owing to the resemblance to hysterical dissociations and the katatonic form of *dementia præcox*. But stupor in encephalitis is rare without physical signs, such as ocular palsies or irritative phenomena or changes in the cerebro-spinal fluid. Cases have been recorded, however, of stupor without neurological signs in persons in whose brains the characteristic reactions were seen at autopsy.

In thirty patients to whose records I have recently had access, and fifteen whom I have seen in the past eight years, the outstanding mental states were as follows:

Lethargy	26
Lethargy with delirium	6
Delirium (including two with the occupational type and three with crises of anxiety or fear)	9
No definite mental change	4
	45

There are special points of interest in the following cases.

CASE III.—A female, aged thirty-six, for years had been of a nervous disposition. For the past two years she had had a constant feeling of being worried without cause. For the past three months she had had a sense of deep depression with considerable difficulty in keeping awake by day. For a month before admission to a clinic she had been very sleepy by day and restless by night and at times aurally hallucinated. She complained of a tired, worn-out feeling, deep depression, insomnia and pains all over the head. Her memory was defective for recent events. She became more somnolent. The main physical signs were general hyperæsthesia and an indefinite plantar response. The optic discs were clear. She died.

Note the history of onset suggesting mental disorder.

CASE IV.—A female, aged forty-three, had been of a happy disposition before an attack of influenza six months before admission to a clinic. Two months after the acute illness she complained of attacks of excited feelings in her stomach during which she felt suicidal. A month after admission she became querulous and then lethargic, with incontinence, diplopia, nystagmus and other physical signs. She improved in the course of a year, but at the present time, more than four years after the acute stage, is reported as complaining of numerous pains and as being unable to work.

Note the mental onset, with emotional attacks and recrudescence of acute symptoms.

CASE V.—A female, aged thirty-six, was strong and happy up to six months before admission; she became nervous, afraid to go out alone, then lost all energy and lay in bed for some weeks and could with difficulty be persuaded to eat and wash. On admission she was lethargic and unable to give much account of herself; there was some head retraction. She improved and became brighter, then complained of pain at the back of the head, became comatose and died a year after onset.

Note the history suggesting a psychosis, with development of definite physical signs only after admission to a clinic and prolonged subacute course.

CASE VI.—A male, aged forty, had loss of appetite and headache for a week and was drowsy and stupid for three days before admission. He was very dull, unable to give a history and was irritable. A few days after admission he talked incessantly and disconnectedly and displayed severe loss of memory even for his wife and family and home address. He had mild pyrexia and various physical signs. No Wassermann reaction was elicited with his blood and cerebro-spinal fluid, but there was a great increase of small cells in the latter. At the present time, a year after his discharge from hospital, his wife states that some days he is too weak to do anything and is almost mute, while at other times he is restless and talks incessantly. He is very forgetful and cannot be trusted out alone. He will jump up suddenly and say that he must get ready to go to work. He declares that he was put in hospital for a day or two "just for a joke." Recently he said to me: "I thought it was all a joke that I was away from work so long and only just now I have woken up to the fact." He has suffered a very considerable degree of

intellectual impairment and his memory for both recent and remote events is grossly defective. His emotional tone varies between moderate euphoria and mild depression with irritability. Beyond sluggish pupils and lingual and digital tremors there are no physical signs.

Note that this patient presents the clinical picture of presbyophrenia. I am under the impression that such a degree of mental impairment is uncommon after epidemic encephalitis. I have the notes of another patient, a man of fifty-two, who also has a profound loss of memory following the acute stage.

The Korsakow syndrome with confabulation and illusions of recognition has been recorded as an unusual occurrence in encephalitis. We have yet to learn how many so far unexplained states of excitement and confusion have a toxic or infective basis, such as epidemic encephalitis.

The Mental Sequelæ of Encephalitis.

Kirby and Davis[2] classified the mental symptoms of epidemic encephalitis under the following heads:

1. Organic reactions, characterized essentially by impairment of apprehension, interference with elaboration of impressions, defects in orientation and retention, difficulty in activization of memories with variability in mental capacity and level of attention, the so-called mental tension defect. It is now well established that this reaction is seen only when the brain has been damaged temporarily or permanently, including injury by toxic infectious agents.

2. Affective reactions, characterized by emotional disturbance following essentially the pattern of the normal affective states. Typical examples are the manic-depressive oscillations and other benign emotional disorders.

3. Trend reactions, characterized by abnormal trend and ideas, an unusual attitude towards the outside world, with constitutional peculiarities and psychogenic mechanisms in the foreground. Examples are schizophrenic and paranoid states.

In their experience the most striking feature in the mental symptomatology of the chronic stage is the change in the emotional reactions which they considered to be due to an inability to show affect rather than to experience emotion.

Mapother[1] in a review of forty-five post-encephalitics found that there were three outstanding types of symptoms, morbid restlessness, moral changes occurring mainly in children, and mental anergia ranging from difficulty in sustaining attention to morbid sleep, occurring mainly in adults.

A. Parsons[3] found that of two hundred and fifty persons suffering from post-encephalitic sequelæ one hundred and thirty-six manifested mental changes as follows:

Impairment	57
Psychic disturbances	47
Drowsiness	15
Neurasthenia	7
Loss of memory	6
Melancholia	4
	136

Gardner Robb[4] examined one hundred and forty-one patients two and a half years after the acute stage and found that 8·8% were well, 7·0% had mild nervous symptoms, 12·0% had a changed character, 13·4% had mental symptoms and six were in mental hospital, 58·0% suffered from Parkinsonism, while 0·8% had other neurological sequelæ.

C. M. Smith[5] followed up one hundred and twenty-eight patients a year after they had been in hospital with acute encephalitis. Of thirty-five children between seven and fourteen years of age nineteen had become restless and naughty; one or two of these seemed precocious rather than mentally retarded. In the whole group of one hundred and twenty-eight there were noted the following:

Insomnia	55
Asthenia	46
Drowsiness by day	31
Psychic changes	24
Nocturnal restlessness	21
Neurasthenia	6
Absent mindedness	3

Insomnia and asthenia appeared most frequently in patients who had otherwise slight physical disabilities and were still able to follow their usual occupations. But since asthenia is so often complained of early in the development of the Parkinsonian syndrome, one would like to know the states of these patients two, three or more years later.

I regret that I am unable to give any figures based on personal experience regarding the frequency of mental sequelæ in encephalitis. I have been impressed by the fact that adult patients who come to psychiatric clinics with a history of acute illness suggestive of encephalitis nearly all have the Parkinsonian syndrome. In the Broughton Hall series there is one patient only in whom mental symptoms dated from an attack of encephalitis eighteen months previously and who displayed no physical signs; he had neurasthenic symptoms, but seemed otherwise healthy. So far as my experience with adults goes, therefore, I must confine myself almost exclusively to the mental accompaniments of Parkinsonism. In patients with post-encephalitic Parkinsonism treated in the wards of the Royal Prince Alfred Hospital and at Broughton Hall the associated mental states were as follows:

TABLE I.

Mental State.	Royal Prince Alfred Hospital.	Broughton Hall.
No special mental symptoms	15	5
Emotional, anxious, excitable	6	3
Depressed	4	9
Dull, lacking in emotion	4	8
Memory poor, intelligence diminished	1	—
Neurasthenic	1	4
Psychotic	—	3
Restless, undisciplined	—	3
TOTAL NUMBER	31	35

It will be seen, therefore, that there is a distinct mental change in 50% of the hospital patients and

in 85% of those referred to a psychiatric clinic. The same patient may pass through a number of stages, from the neurasthenic to the depressed and perhaps to the permanent stage of emotional dulness with more or less intellectual impairment.

CASE VII.—A man of twenty-eight years had had an influenzal attack with drowsiness and diplopia four years previously. Ever since the acute stage he has felt unwell, easily fatigued and lacking in energy. There was a gradual development of cerebellar symptoms. A few weeks before admission to a clinic he attempted to cut his throat after being told that his condition was incurable; he remained very depressed for a time. He is severely incapacitated from incoordination, but has become resigned and fatalistic. There is no intellectual impairment beyond impaired concentration and he tries hard to cooperate in any treatment which is suggested.

I feel sure that attempts to regard any single mental symptom as a fixed reaction type are likely to be misleading, unless the patient is observed over a long period of time. A case quoted by Duncan[6] illustrates this point.

A girl of fourteen recovered from the acute stage with the residual symptoms of unequal pupils and exaggerated knee jerks, but remained very apathetic. A few months later she was admitted to a mental asylum as a typical case of *dementia præcox* and her condition improved until three years after the onset of the illness, when she became restless, noisy and delusional and after a period of excitement she passed through a dull, stuporose phase and finally became bright and intelligent. For the past ten months she has been apparently normal.

Taking the Broughton Hall series in greater detail, there are several points worthy of note. Although comparison with a series of patients in a general hospital appears to indicate that mental symptoms are more prominent in the Broughton Hall patients, there is surprisingly little evidence of psychopathy previous to the encephalitis. Two patients were dunces at school and one of these showed a mental reaction which is quite in keeping with an inferior mentality.

CASE VIII.—A man of twenty-two years had been very backward at school. He had had an acute illness four years previously and had been delirious, after which he had remained fairly well for two years. He had then become emotional and pannicky, forsook his work to attend revival meetings and complained that people had a "down" on him. At about the same time he complained of stiffness in his arms. He was also difficult to manage at home. In the clinic he was foolish and irresponsible, asked trivial questions and often cried without being able to give a reason. He somnambulated on several occasions and once climbed on to a veranda roof at night. He had an intelligence quotient of 70. The Parkinsonian syndrome was present.

Two patients had been of a nervous disposition, were easily frightened and excitable for years and one of these had emotional crises after encephalitis. One may therefore conclude that there is very little correlation between the temperament and emotional qualities before and after the illness.

A history of acute illness, influenzal in type, with delirium, somnolence, diplopia, hiccup or some other accompaniment suggestive of encephalitis was obtained in thirty-one out of the thirty-five patients.

Development of Chronic Symptoms After the Acute Stage.

Fifteen patients have not been well after recovery from the acute stage. Of sixteen others three had symptoms six months after the acute illness, two had symptoms one year after, four had symptoms two years later, one had symptoms three years later and six had symptoms four or more years after the acute illness. There was no history of an acute illness of four patients.

The first symptoms were neurasthenic (fatigue, weakness) in sixteen, depression in six, increasing slowness and stiffness in six, tremors in five.

The diagnosis may be difficult in the early stages of Parkinsonism owing to resemblance to neurasthenia or melancholia. The following case is instructive.

CASE IX.—A man of thirty-eight years complained that for several months he had become increasingly fatigued, had lost interest in life, felt depressed and reproached himself for trivialities. He stated that in the early twenties of his life he had felt somewhat as he did at present and had then been so depressed that he was treated in a mental hospital for over a year. He had made a good recovery. He had served overseas during the Great War and had had several attacks of malaria which had left him run down and mildly depressed. He had also had influenza in 1919 after which he had remained well for five or six years until his present illness. In view of his history and of the absence of any special physical signs I diagnosed recurrent melancholia and was very surprised some three or four months later to observe unmistakable signs of Parkinsonism, with manual tremors, salivation and the characteristic mask, gait and attitude. With the development of these signs he became unemotional rather than depressed.

I think that many cases can be diagnosed only after prolonged observation. The patient with well developed melancholia displays the furrowing and wrinkling of depression rather than the fixed and smooth features of the Parkinsonian mask, nor are the eyes so staring and prominent. Salivation is absent. Rigidity may or may not be present in the melancholic and is not so much passive as resistive and active. The emotional tone and ideation are definitely depressive. The Parkinsonian, on the other hand, in addition to his attitude and conative impediments nearly always presents physical signs of a type not found in melancholia, particularly defects in the movements of the eyeballs and in the reactions of the pupils. Nevertheless, it is possible that some patients classed as chronic melancholics in mental hospitals may be found to have mild signs of this organic syndrome.

The katatonia of *dementia præcox* may cause difficulty. But one must not be deceived by mere similarity in attitude. Katatonia is associated with a number of quite distinctive mental symptoms, with bizarre utterances and conduct and is a disorder of personality. Here again the katatonic attitude is an active resistance to external interference, a means of avoiding contact with the human environment. It is true that in katatonia as in other mental disorders certain primitive mechanisms may be released and activated owing to the suspended activity of the higher controlling levels. Hence the similarity between some functional and organic conditions.

I have already indicated that the Parkinsonian syndrome may develope through a prolonged neurasthenic phase, in which unusual fatigue and sense

of weariness, often with impaired concentration, constitute the main symptoms and which usually persist when the physical signs have become definite. In some instances these symptoms persist with considerable intensity, while physical signs fail either to develope or to reach a severe degree—the so-called asthenic syndrome. The resemblance to neurasthenia of mental origin is so close that one may spend much time in searching for possible psychological factors. Careful and repeated examination should be made for defective pupillary reactions and impaired conjugate movements of the eyeballs, for slight facial palsies and for tremors.

Precipitating Factors.

In the great majority of cases the onset of the Parkinsonian syndrome is insidious and in my experience is rarely ascribed by the patient to any special circumstance. In a few instances a melancholic self-depreciation may lead the patient to ascribe his condition to the sins of the past; such conditions are referred to in another section. In the following case the physical condition was ascribed by the patient to trauma.

CASE X.—A male, aged thirty-five years, had "pneumonic influenza" in 1919, was drowsy and delirious and tried to strangle his wife; he also had diplopia. He returned to work six months later, but on the third day he received an electric shock and developed severe pains in the left side and in the head, lost his power of concentration and became weak and fatigued. A year or so later the Parkinsonian syndrome was well established and was ascribed to the shock.

There is a danger of being misled by apparent psychogenic factors.

CASE XI.—A female, aged thirty-five years, attended a clinic with the statement that she believed that she had had an abortion some months previously. She had felt very ashamed at the time and afraid that her family might get to know about it and had adopted a rigid attitude to restrain any further hæmorrhage. She reproached herself for illicit sexual relations and explained that she now felt unable to relax from her rigid pose. She presented a typical picture of Parkinsonism and closely resembled a waxen doll. She could stand for long periods with her arms moulded into unusual positions without seeming to tire. There was no history of any acute illness suggesting encephalitis. The doctor who had referred her to the clinic, had been sufficiently impressed by her story to diagnose hysteria and request that she receive psychotherapy. Hyoscine gave her some relief for a time, but her physical condition became worse. The truth of her story was never ascertained as the details were too vague.

Psychoses after Encephalitis.

It is remarkable that in spite of the varied mental changes which may follow encephalitis, in very few individuals does the disorder of conduct lead to certification. Early in 1925 in England there were only 210 post-encephalitics under certificate, half of whom were under the age of twenty. I am much obliged to Dr. Hogg, the Inspector-General of Mental Hospitals, for the information that during the past ten years only nine patients with acute encephalitis have been admitted to the New South Wales mental hospitals, five of whom were voluntary patients in Broughton Hall. In sixty-five the mental symptoms have been attributed to the effects of encephalitis and of these no less than forty were in Broughton Hall.

Sometimes one sees deep depressions, typically melancholic in their mental symptomatology, occurring in Parkinsonians and passing off again.

CASE XII.—A female, aged twenty-seven years, had been of a happy disposition. She had influenza three months ago and saw double. Ever since she has felt sleepy and depressed and has noticed that her movements have become slower. She became acutely depressed and full of self-reproach, blaming pre-marital intercourse for her present condition. Her mental state was typically melancholic with retarded ideation. There was moderately severe Parkinsonism. Mental but not physical improvement took place.

I have met with few Parkinsonians who have expressed any deep sense of hopelessness or who have declared that they were better dead. One patient in the Broughton Hall series committed suicide in another institution.

CASE XIII.—A female, aged forty-four years, was said to have been unhappy before marriage and to have quarrelled with her younger sisters. Her married life was happy, but she remained on bad terms with her own relations. She had severe influenza in 1919 and was delirious, after which she remained well for four years, when she noticed that her movements were becoming slow and stiff. A little later she complained that her sister had put powdered glass in her food and also expressed ideas of religious persecution. She declared that the stiffness in her arms was due to poisoning and electricity. She was frequently noisy at night and called for help; at other times she described her persecutions with a smile. She had a moderate degree of Parkinsonism and usually sat immobile with fixed gaze, but occasionally became aggressive and violent. She was sent to a mental hospital.

Note the mental symptoms of the paraphrenic or *dementia paranoides* type apparently precipited by the physical disability. Persecutory ideas were at first against those with whom she had been quarrelling for years.

Emotional Crises.

Several of the patients displayed emotional crises amongst other symptoms.

CASE XIV.—A female, aged thirty-seven years, had not felt well since a severe attack of influenza three years previously. She is mentally dulled and usually lacks emotional expression. For brief periods she becomes restless, mildly excited and breaks out into a profuse perspiration. Choreiform movements constitute the main physical signs.

CASE XV.—A female, aged thirty-three, years, has a Parkinsonian syndrome of about a year's duration. There is no history of an acute illness. She is querulous and mildly depressed. At times she has what she terms a very unpleasant feeling and she becomes very emotional, at the same time there occurs a spasm of the ocular muscles, so that the eyes become fixed in an upward direction.

As Wimmer and others have pointed out, these ocular spasms are liable to occur when the patient is under the influence of a strong emotion. But in my experience the ocular spasms and the emotional crises have often appeared to occur simultaneously, without the patient being aware of any precipitating factors, "the feeling comes over her." One must, therefore, look for causes within the vegetative nervous apparatus.

To summarize, the outstanding symptoms in the chronic stage in adults are abnormal fatiguability, impaired concentration and most of all the retarded ideation which has been given the name of bradyphrenia (Naville)[7] corresponding to the bradykinesia of the Parkinsonian syndrome with which it is so frequently associated. The emotional tone takes the form of dulness and indifference, but there may be deep depression. Judgement is but little impaired in most cases, but the patient is unable to exert his mental processes as well as before the illness. Many patients appear surprisingly indifferent to their condition and even cheerful, attitudes which may be regarded as merciful dispensations in such a crippling disorder. One is reminded of the *spes sclerotica* and eutonia described by Cottrell and Wilson[8] in multiple sclerosis. Some authors, notably Kirby and Davis, have considered the emotional loss in post-encephalitic states to be due to an inability to display emotion, but I have been impressed with the lack of concern and fatalism of many of my patients who, unlike melancholics, do not worry over the defect of emotional expression.

Treatment.

The treatment of the acute stage lies within the province of general medicine. I have already indicated that removal to a mental hospital is necessary only in the rarest instances. Special observation may be called for when there is deep depression with suicidal tendencies.

The psychiatrist sees more of the sequelæ, especially of the Parkinsonian syndrome, either because of the associated disorders of mood and conduct or because in its mild and developing form this syndrome has been mistaken for neurasthenia or some other mental disorder. In addition to medicinal therapy, hyoscine and the like, which in some instances afford the patient mental relief in so far as the rigidity is reduced, the *régime* of ordered occupation and recreation offered by a psychiatric clinic infuses a certain measure of cheerfulness into those who have become discouraged and depressed. The learning of a simple handicraft gives the Parkinsonian a new interest in life when he has cause to look upon himself as incapable of anything. In a few there is improvement in severe depression independently of any change in the physical condition. On the other hand, emotional apathy saves many of those who are most severely handicapped from useless introspection and concern for the future. It must be admitted that the therapeutic possibilities for the majority of patients are poor, especially as the virus may lie latent for prolonged periods and then cause a new destructive process.

Encephalitis in Children.

The acute stage in children presents no special features. Drowsiness, delirium and great excitement occur as in adults. These features were all noted by Cleland and Campbell[9] when the epidemic reached Australia in 1917. The young organism with its plastic and still developing nervous system is relatively more affected than the adult, hence the greater liability to mental arrest or impairment and the strange alteration in disposition which so frequently takes the form of anti-social behaviour. In contrast to adults children and adolescents seem more liable to develope mental without physical sequelæ. Hall[10] has classified the clinical types in children as follows: Idiots, Parkinsonians, apaches, difficult children.

Idiocy or mental arrest of varying degrees appears more likely to occur in infancy or early childhood. It was, of course, well recognized before the epidemic that the different forms of meningitis, especially in association with the exanthemata, might be followed by mental defect. During the school years also mental impairment may follow encephalitis, but more often the failure to maintain average progress is due to a lessened power of concentration and an abnormal fatigability combined with a slowing of performance. Shrubsall[11] found that many of his patients who appeared to be mentally dull, could pass the Binet-Simon tests for their age, if urged and allowed ample time.

The Parkinsonian syndrome is rarely seen before the age of ten.

In regard to the apache and difficult types, many children who have been clever and well behaved before their illness, afterwards become dirty in their habits, dishonest, untruthful, quarrelsome and undisciplined and may even display homicidal and suicidal impulses. Auden[12] followed up patients with epidemic encephalitis and cerebro-spinal meningitis living in the same district over a period of three years and found a number with conduct disturbances after the former, but none after the latter. He summarized the changes in these words:

> Increased emotional instability, with a reduction in the volitional inhibitions, leading to aberrations of conduct, a marked restlessness, especially in the evening, accompanied by nocturnal wakefulness (with which may be associated hallucinatory manifestations) an apathy or inability for sustained attention and an irregular type of intelligence capacity as revealed by educational tests. The majority of these cases shows heightened sensibility to environmental stresses.

Shrubsall[11] studied 241 cases of post-encephalitic children under the age of sixteen during 1926. Of these seventy-eight appeared to be normal and without sequelæ and twenty without any definite signs were described as "not so well as before their illness." The remaining 143 manifested the following sequelæ: Sleep disturbances 41, Parkinsonism 10, excito-motor changes 20, paralyses of various kinds 28, impaired intelligence 56, disorders of conduct 70.

In seventy children with disordered conduct after encephalitis Shrubsall found irritability, morbidness, restlessness and excitability in forty-five, lack of control in twenty-six, conversion neurosis in four, pilfering in eighteen and sexual assaults in two.

The change in disposition involving a weakening of the moral sense is the most striking sequel in children. There may be a certain lack of restraint,

a forwardness and cheekiness which is quite foreign to the child's former self. He begins to accost and pester strangers in the street and annoys his elders in petty ways. He may play truant at school, especially when an inability to concentrate and a sluggish ideation lead to failure in keeping up to the average. Wanton destructiveness, stealing and sexual misconduct are some of the more serious developments.

CASE XVI.—A boy, aged seven years, remained restless after an attack of encephalitis. He was admitted to a clinic, but was difficult to control and made himself a nuisance to the older patients. He complained to his parents that a male attendant had come into his single room at night and exposed himself. The boy later gave a disconnected and sketchy account of the supposed episode and failed to supply essential details. He also stated that he saw faces at the window. Evidence obtained from other patients and from a patrolling night sister showed the boy's story to lack foundation.

The following case exemplifies the delinquent trends which may follow encephalitis.

CASE XVII.—A boy of eighteen years was well behaved and made steady progress at school up to the age of twelve, when he had epidemic encephalitis and was treated in a hospital for six weeks. After this illness he quarrelled with his teacher and refused to submit to rules and so was sent away from school. A year or two later he was convicted of stealing and sent to an industrial school where he caused a good deal of trouble. Since then he has had several convictions. His thefts are petty and although he admits that he hardly steals to his advantage and is usually caught, yet he cannot control the impulse to take things which attract him for the time being. He is of average intelligence and does not present any physical signs. He seems likely to repeat his offences.

The following illustrates the post-encephalitic change in character.

CASE XVIII.—A male, aged nineteen years, whose mother is nervous and moody, was healthy and happy as a child and displayed no psychopathic traits up to the age of fourteen. He had made average progress at school and obtained a certificate. Soon after he had a severe attack of influenza and felt dizzy for some time. He then became sleepy and indolent and complained of a lack of energy. He ascribed his condition to self-abuse and his father threatened him with severe punishment. He failed to settle down to steady work and tried several kinds of occupation without success. His parents reported that he took to lying without apparent motive and that he had become most insolent. His father had thrashed him several times for his laziness. At the age of nineteen he complained that his movements had become slow and stiff and that he frequently got the "trembles." He has the Parkinsonian syndrome and while in a clinic was a source of annoyance to other patients and did not improve.

Unsympathetic handling and an inability to satisfy the demands made upon them may be a determining factor in the development of disobedience and aggressiveness.

CASE XIX.—A boy of seventeen years came under my notice in London. His doctor stated that he had become a disciplinary problem at home, that he was subject to violent outbursts of temper and that he was lazy and indifferent about his future. The diagnosis of *dementia præcox* had been suggested. The boy's father confirmed these statements and admitted that he had lost all patience with his son who had become a good-for-nothing during the past two or three years. The boy then walked in and there was something about him that suggested the Parkinsonian gait, although even after watching him carefully I could not persuade myself that the diagnosis was beyond question. He then sat down and after describing his symptoms quite alertly for a minute or so suddenly fell asleep. He had already volunteered the information that he had felt his movements becoming stiff, that he had attacks of sleepiness by day and that saliva sometimes dropped out of the corner of his mouth. There was no history of an acute stage. The boy admitted being extremely irritable when his father urged him to activity. The case was explained to the father and hyoscine prescribed by the mouth. A few weeks later I was informed that harmony had been restored at home, that the boy seemed brighter, but that the Parkinsonism had not improved.

It has been suggested that the undisciplined conduct results from faulty parental control after the acute stage when a lack of understanding is countered by the patient by irritability and the desire to get his own back. These behaviour disorders constitute a problem which still lacks a satisfactory solution. Although ordinary punishment is of no avail and may lead even to more severe misconduct, the ordered routine of institutional life provides a wholesome corrective to antisocial trends and deprives the adolescent of the prominence into which his conduct brings him at home. Industrial and farm colonies appear to supply the best environment for adolescents. Education should be carried on as far as possible during the school years and in large cities special classes may be formed for retarded children. Should misconduct persist, recourse must be had to a complete change of environment and education may have to take a second place.

In conclusion I wish to thank the members of the honorary staff of the Royal Prince Alfred Hospital for access to the records. I wish also to express my special indebtedness to Dr. Evan Jones for the comprehensive records which he placed at my disposal at Broughton Hall.

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THE BILIARY TRACT: RECENT ADVANCES IN KNOWLEDGE.[1]

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PROBABLY no department of medical science has attracted more attention in recent years than the physiology, pathology and surgery of the biliary tract. The subject has been studied from every angle; interest has been maintained by important discoveries and curiosity stimulated by the pleasure of puzzles about to be solved and mysteries elucidated.

It is not easy in the profusion of writings that have appeared, to separate fact from accepted theory. Rational views may be widely divergent and one might say, in fact, of the gall bladder that its demonstrated functions are important, but on the other hand it has been proved to be a useless encumbrance.

Too often it renders burdensome that period of life when we look for rest and comfort and it is well, perhaps, that man may continue to live happily though deprived of his gall bladder.

The structure of the gall bladder is simple, consisting of four layers: columnar epithelium, fibromuscular layer, subserous layer of connective tissue and an incomplete serous layer. It has a generous vascular system and a rich lymph supply. The lymphatics communicate freely with those of the first part of the duodenum and liver and the head of the pancreas.

The Bile.

In health the bile consists principally of water, bilirubin, salts of the bile acids, free cholesterol and mucin. Bilirubin is derived from hæmoglobin It is an iron-free derivative of hæmatin and the amount of its production depends on the amount of blood destruction, as is illustrated in cases of acholuric jaundice in which the jaundice is due to the fact that more bilirubin is produced than the liver is able to excrete. The formation of bilirubin from hæmoglobin is undoubtedly an important process, for by it iron is conserved and not lost to the body.

Much experimental work has been done to determine the site of bilirubin formation and this appears to have been definitely established. It is one of the functions of certain specialized endothelial cells found in the spleen, bone marrow and liver.

Of a number of tests for bilirubin the Van den Bergh has been most used. It has been of sufficient accuracy for a good deal of the experimental work and is also of great use and ready clinical application. Recently, however, Sheard, a physicist in the Mayo team, has introduced a very delicate spectrophotometric method by which bilirubin can be estimated with great accuracy. The limit of the Van den Bergh test is about one in 100,000 or one in 200,000. Sheard's method is about fifty times more delicate. Using this method, Mann, also of the Mayo team, has estimated the bilirubin in the splenic and tibial circulations. He found by injecting hæmoglobin into the artery that a rise of bilirubin content occurred in the venous blood. By other ingenious experiments he showed that bilirubin was also formed in the liver.

The fate of bilirubin is excretion. It is not reabsorbed from the intestine. It is a waste product. It would not attract so much attention to itself were it not for two properties that always compel notice —a vivid colour and an affinity. The colour is that of jaundice and the affinity is calcium.

Of the bile salts not much can be said. There is so far no reliable method of estimating them. It is known that they strongly stimulate the secretion of bile and it is thought that they help to keep the cholesterol of the bile in solution or emulsion. Cholesterol is a lipoid of the group containing neither nitrogen nor phosphorus. The site of its production in the body is not known. It is normally present in the circulation as an ester. The polygonal cells of the liver are presumed to deesterize it and it appears in the bile as pure cholesterol. From the bile it is reabsorbed by the gall bladder and possibly also by the intestine. It has been thought that cholesterol metabolism is conservative, like that of iron and that none is lost to the body. It is also said that more is excreted as coprosterol than is derived normally from food.

Experimentally it has been proved that the gall bladder can absorb fat from bile and Mentzer has satisfied himself experimentally that the same is true of cholesterol. He used a solution of cholesterol crystals in oleic acid. The result was not conclusive as in the case of the easily recognized Sudan-stained fat. In man it is found that cholesterol does not accumulate in the gall bladder wall unless the cystic duct is patent and in laboratory animals ligature of the cystic duct prevents its appearance in the tissues of the gall bladder. The inference is that cholesterol found in the wall of the gall bladder is derived from the bile and not from the blood stream.

Functions of the Gall Bladder.

The healthy gall bladder can concentrate its content of bile by the absorption of water. This has been easy of proof in the dog in which, owing to the arrangement of the biliary system, bile may be drawn separately from one of the bile ducts and from the gall bladder.

In man the appearances found radiologically after the administration of sodium iodophthalein indicate the same function.

Of very great interest is another function of the gall bladder, namely the absorption of cholesterol.

A third function of the gall bladder is that of its muscular and elastic layer. It is generally believed that the gall bladder can contract and empty or partially empty its content into the duodenum. Mentzer enunciated a theory of "contrary innervation" of the gall bladder and the sphincter of the common bile duct. The sphincter relaxes as the gall

[1] Read at a meeting of the Queensland Branch of the British Medical Association on March 2, 1928.

bladder contracts and so bile is expelled. The theory is at present not capable of either proof or disproof, but is accepted as analogous with a similar function in other hollow viscera. Emptying of the gall bladder occurs when a meal, containing fat or protein, is discharged into the duodenum. A concentrated solution of magnesium sulphate has been shown to produce the same result.

The bile in the gall bladder is generally under a slight pressure, about 150 millimetres of water in the dog. The pressure in the common duct is not always the same as that in the gall bladder which indicates that there may be some other factor concerned in the emptying of the gall bladder besides the contraction of the gall bladder and relaxation of the sphincter of the common duct. Graham and Kodama have been at some pains to prove that the gall bladder does not actively contract, but Mann, in observing the emptying of the gall bladder in the dog, has noted rhythmical alterations of pressure of about forty millimetres of water occurring once to five times a minute.

In animals not cursed with a gall bladder the sphincter of the common duct is functionless and the pressure in the biliary tract is not more than about thirty or forty millimetres of water. The same condition is produced artificially by cholecystectomy and in fact the success of that operation, when undertaken for gall bladder disease, depends on the subsequent loss of tone of the sphincter.

Cholesterosis and Gall Stones.

Twenty years or more ago Moynihan described the condition which is now generally called strawberry gall bladder. The appearance is due to deposits of cholesterol scattered over the dark red inflamed mucosa. These deposits may increase in size and form villous projections or papillomata. The condition is associated with an increase of cholesterol in the circulation and is probably due to that excess. Hypercholesterolæmia gives rise to an increase of cholesterol in the bile and from the bile it is absorbed by and accumulates in the gall bladder. If the cystic duct be not patent, cholesterol will not be found in the gall bladder tissues.

This disturbed cholesterol metabolism is related to the formation of gall stones in a large percentage of cases. Nearly all young stones are cholesterin or cholesterin rich. Moynihan and many others have found a hypercholesterolæmia in these early cases. At a later stage there may be a normal or decreased cholesterolæmia which is to be explained by the loss of cholesterol absorption by the diseased gall bladder. In these late cases the patients have an increase of calcium in the blood bile and gall bladder and calcium is found in the gall stones.

After cholecystectomy the cholesterol content of the blood approaches normal. Presumably when the gall bladder has been removed or has ceased to function, less cholesterol is reabsorbed and more is excreted as coprosterol in the fæces.

What part does infection play in the formation of gall stones? Clinically we hardly think of cholelithiasis apart from infection and yet there is an increasing doubt whether infection has been present in all cases.

Many observers (Churchman, Judd and others) have reported their frequent failure to demonstrate organisms in the gall bladder, even in the presence of gross inflammation, and gall stones have been produced in healthy animals by overfeeding with fat and in the absence of infection. There is no doubt, however, that the majority of diseased gall bladders are or have been infected and a variety of organisms has been found. There are several possible routes of infection. The bile secreted by the liver may contain organisms, but we know that the healthy gall bladder is not easily invaded and normal bile is not a good culture medium. It is a constant experimental finding that the gall bladder is not infected by merely inoculating its contained bile and it has been found that the bile may teem with typhoid bacilli without causing cholecystitis.

There is more to be said in favour of hæmatogenous infection. Rosenow's work is well known. He has shown that streptococci have a selective affinity for certain tissues. Some strains prefer gall bladders, others appendices, others again joints or fibrous tissues. In laboratory animals streptococci from various lesions tend strongly to reproduce the same lesion when injected intravenously. His most recent table of results shows some very interesting points. Certain affinities between lesions are clearly indicated. Thus streptococci from a patient with cholecystitis commonly reproduce cholecystitis and also duodenal ulcer, but seldom appendicitis. Streptococci from a patient with appendicitis reproduce appendicitis and very often also fibrositis and myositis, but very seldom produce cholecystitis.

A third possible route of infection is the lymphatic route. Graham, Starr, Judd and Moynihan have shown that hepatitis is a constant accompaniment of cholecystitis. It is often gross and obvious to the eye and it is always to be found microscopically. There is a free communication between the lymphatics of the gall bladder and of the liver, especially around the bile ducts and bile capillaries. It is these parts that are always found to be infiltrated by leucocytes. Graham points out that in cases of early cholecystitis it is always those areas of the gall bladder and the liver which are rich in lymphatics that are found to be inflamed, namely the subserous areolar tissue of the gall bladder and the connective tissue around the bile ducts and capillaries. The condition has been produced experimentally by injection of organisms into a radicle of the portal vein. In other experiments biliary infection by *Bacillus paratyphosus* has been worked out. The finding was that cholecystitis was invariably accompanied by hepatitis and that bacillary infection of bile could occur without causing cholecystitis.

Jaundice.

We are indebted to McNee for much of our knowledge of jaundice and for the accepted classi-

fication of different forms. We are tonight concerned only with obstructive jaundice, but a summary of present views of the causes of jaundice and its production may be presented.

Jaundice is that condition in which the amount of bilirubin in the circulation is above normal, normal limits being between one in 1,000,000 and one in 400,000. Jaundice becomes obvious to the eye and bile can be detected in the urine when the bilirubinæmia reaches one in 50,000. Between these points the latent jaundice may be detected by the Van den Bergh test.

Jaundice may be brought about in a number of ways. When there is obstruction to the bile flow, the pressure may reach a point at which the polygonal cells, though healthy, can no longer secrete bile. McNee, however, believes that it is secreted and reabsorbed. This is obstructive jaundice. In the absence of obstruction, the polygonal cells may be so damaged that they are unable to perform their excretory function and bile accumulates in the circulation — toxic jaundice. Both conditions, mechanical obstruction and toxæmic damage to the liver cells, may coexist as in catarrhal jaundice.

In hæmolytic jaundice there is, owing to excessive blood destruction an excessive production of bilirubin and jaundice occurs because, although the biliary tract is not obstructed and the polygonal cells healthy, the latter are unable to excrete bilirubin as fast as it is formed.

Obstructive jaundice, then, will occur when the pressure in the biliary tract reaches a certain height and it is clear that this will occur only when the gall bladder has reached the limit of its capacity to concentrate bile by the absorption of water, for owing to this capacity it can store all the bile that is secreted in twenty-four hours or more. In the dog it has been shown that the presence of a healthy gall bladder can delay the occurrence of jaundice for thirty-six to forty-eight hours after the common duct has been ligated. If, however, the cystic duct is also ligated, a rise in the bilirubin content of the blood can be detected by the Van den Bergh method in about three hours. A healthy gall bladder, then, may provide internal biliary drainage for as long as thirty-six or forty-eight hours.

Surgical procedures in jaundiced patients are always fraught with risk. Fortunately in the majority of cases the obstruction is relieved and the biliary flow reestablished without surgical intervention and the operation of choice may be undertaken later at a favourable moment. Sometimes, however, an operation has to be performed for the relief of the obstruction and under these circumstances the risk may be great for two reasons principally, first because there may be a decreased coagulability of the blood and secondly because there may be some degree of hepatic insufficiency owing to damage to the polygonal cells of the liver. Preoperative treatment may greatly diminish both these sources of danger. The decrease of coagulability of the blood is due to the affinity of bilirubin for calcium whereby a portion of the calcium in the blood combines, forming an inert compound which we may call calcium bilirubinate. In its combined molecular form calcium is not available for the process of thrombosis. Only the free calcium ion is of use. It used to be the practice to administer calcium by the mouth under these circumstances, but calcium is not readily absorbed. However, it is found that three daily doses of about 0·5 gramme given intravenously are effective. One cannot foretell without having the clotting time of the blood estimated whether it is much increased or not. The increase does not necessarily occur soon after the appearance of jaundice. It may not occur for some weeks.

As regards hepatic insufficiency, it seems that the immediate danger is a hypoglycæmia which may be met by the free administration of glucose both before and after operation.

In Mann's experiments on dehepatized dogs he found that death would occur in about three hours from hypoglycæmia. By the administration of glucose the animals might be kept alive for thirty-six hours and death was then due to some cause other than hypoglycæmia. To some extent it appeared that the muscle glycogen could be drawn upon to maintain the blood sugar level, but muscle glycogen is not readily available and death occurs before this source of supply is exhausted.

Diagnosis of Biliary Conditions.

Recent advances in diagnostic methods are a considerable triumph. It is as important as ever to obtain the most careful history. There is no better description of the beginnings of gall bladder disease than that of Moynihan. The procedure by which the bile may be drawn off from the ducts and gall bladder, is the work of Lyon. A positive finding of cholecystitis may be made by this method when the bile, identified as coming from the gall bladder, contains inflammatory products and some idea of the degree to which the gall bladder can still concentrate its content of bile, may be formed. Very often, however, no abnormal constituents of the bile are found even in the presence of definite cholecystitis. There may well be pronounced changes in the periphery of the gall bladder with as yet no inflammatory exudate in the bile.

Of more value is the radiological examination after the administration of sodium tetraiodophenolphthalein. This method constitutes a reliable test of two of the functions of the gall bladder, namely, its capacity to concentrate bile and its capacity to empty itself after a meal. The diagnosis made by this method alone is right in more than 90% of cases.

We have all been confronted with the problems of differential diagnosis as between cholecystitis, peptic ulcer, appendicitis and pyelitis *et cetera*, but there is one condition that is frequently overlooked and is common enough to warrant more notice. It is colonic spasm.

In chronic spastic constipation there are certain parts of the colon which are subject to painful spasm with dilatation proximal to the spasm. One common situation is the proximal portion of the

transverse colon. Ballooning of the ascending colon results and the pain and tenderness may be located in the right hypochondrium. The condition may be associated with chronic appendicitis, but is thought to be more often due to the prolonged use of aperients, a coarse diet, enemata and the methods usually employed to relieve constipation. The clinical picture may simulate cholecystitis the more so because it is persistent and chronic with occasional acute exacerbations. No doubt some of these patients are suffering from true chronic appendicitis. At any rate the history gives a clue to the diagnosis which is readily established by a careful X ray examination; this should, if possible, include a screening during an attack of pain.

In diagnosis Hurst emphasizes the importance of palpation of a tender visualized gall bladder. However, a diseased gall bladder is frequently not visualized at all and the most tender organ may be the liver.

Treatment of Biliary Conditions.

It is when we come to consider treatment from a theoretical point of view, that we realize how much remains to be learned of disease of the biliary tract. We do not yet know the origin of cholesterol and we are ignorant of the nature of that disturbance of cholesterin metabolism which is commonly present. Further, we do not certainly know the route of infection of the gall bladder.

Hurst and others claim that the infected gall bladder may be sterilized by the administration of very large doses of hexamine, enough alkali being given to keep the urine alkaline and to prevent unpleasant urinary symptoms. Hexamine is apparently active in alkaline bile, if infected, owing to isolated areas of acid production brought about by colonies of microorganisms.

Drainage of the gall bladder may be promoted by instillation of a concentrated solution of sulphate of magnesia through a duodenal tube or by oral administration, the stomach being empty.

On theoretical grounds not much can be expected from this treatment, for it is generally the subserous layer of the gall bladder which is first affected and sulphate of magnesia does not empty the gall bladder more effectively than a meal containing protein and fat which can very conveniently be administered three times or more daily.

No doubt much work is being done with the various dyes which are excreted by the liver, many of which are strongly antiseptic and some of which are absorbed by the gall bladder wall. Mann has investigated mercurochrome and has found it in the bile in sufficient quantities for bactericidal purposes as determined with bile *in vitro*, but he found that the dye as excreted was inactive and he presumed that it was present in a combined form, having been altered in its passage through the liver.

The Results of Surgery.

From the literature it is gathered that the chance of a good result after cholecystostomy is about 40%, of a moderately good result 45% and of a bad result 5%. After cholecystectomy the chances of a good result are about 70%, of a moderately good result about 20% and a frankly bad result 10%.

Starr Judd has followed up several hundred patients with gall bladder disease treated by cholecystectomy in an endeavour to correlate results with the clinical and pathological picture and account for indifferent and bad results. He found in a general way that the more severe the condition, the greater was the prospect of relief after cholecystectomy. Thus in mild early cholecystitis and cholesterosis without stones there were good results in about 70%, moderately good in about 20% and bad in 10%. In the more severe cases, for example, strawberry gall bladder with gall stones, the results were good in 90% and bad in only 2%. Those patients whose illness was characterized by biliary colic and who sought relief on that account, were the ones most certain to be benefited.

From what we know of gall bladder disease, it is clear that cholecystitis and cholesterosis are only part of the clinical picture. Hepatitis is always present and may be the antecedent condition. There is frequently other disease in the portal area. Neglect to treat associated achlorhydria (about 20% of cases); hypochlorhydria (about 20% of cases) or hyperchlorhydria may account for some of the indifferent results. There is still, however, a proportion of cases in which biliary colic persists. Some of these may be due to chronic pancreatitis and a few to stones in the ducts overlooked at operation. It must be borne in mind that the success of cholecystectomy depends on the fact that the sphincter of Oddi becomes relaxed. May it not be that this little sphincter sometimes remains spastic so that the colicky pains of biliary obstruction continue?

Anastomosis of the gall bladder with the stomach or duodenum may sometimes be the operation of choice. This operation provides an alternative internal drainage. Unfortunately the internal fistula frequently closes, if the common duct remains patent or only intermittently obstructed as in pancreatitis.

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THE NORMAL AND ABNORMAL APPENDIX AS SEEN BY THE RADIOLOGIST.[1]

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BEFORE starting on my subject I wish to mention that in this paper I will not refer to acute inflammations of the appendix, as this condition practically never comes under the professional notice of the radiologist, but only to chronic pathological conditions of that organ. Many patients are referred to him for examination of their gastro-intestinal tracts with symptoms of dyspepsia, chronic abdominal discomfort or pain, flatulence *et cetera* and with a provisional diagnosis of chronic gastric or duodenal ulcer or chronic appendicitis.

In all such cases a complete examination is necessary and it may be several days before that examination can be completed and it would help the examiner very much if those advising their patients to have the examination, would tell them that it may take such a time, so that the patients will not get impatient and feel that they are being "strung on" unnecessarily. The examination cannot be hastened and the patient should be seen at least once a day while the opaque meal traverses the whole tract, even if a gastric or duodenal ulcer is revealed beyond doubt, for there is to my mind a not infrequent association of chronic appendiceal pathological change with chronic duodenal ulcer.

Technique of the Examination.

As regards the technique of the examination, I use a barium meal, maizena, cocoa, milk and with sugar to taste and use no special methods when examining the appendiceal region. In a large portion of cases the appendix can be seen and examined on the screen and shown on films.

I think it is wise to avoid all purges and aperients for forty-eight hours before commencing the examination, as they are apt to upset the usual routine of the digestive tract for some time and it is necessary to see the bowel under as normal conditions as possible.

The times of observations have to be varied according to the patient, but as a rule I see patients from five to six hours after the first meal, then four hours later, at twenty-four hours and thereafter every day till the examination is complete.

The Normal Appendix.

With regard to the filling and emptying of the normal appendix some extraordinary statements have been made. It has been stated by one man that the opaque meal never enters the lumen of a normal appendix and that if it does enter, the appendix is pathological; while another holds to the contrary that if an appendix does not fill with the opaque meal, it is abnormal. Such dogmatic statements I think are very harmful. The normal appendix undoubtedly fills and empties freely; such a one is frequently seen, of normal size, shape and calibre, appearing on the screen to float freely in the abdomen. It is movable, not tender on pressure and occupies different positions at different times. It is sometimes difficult to be sure that the tip is not fixed on palpating it, especially if it is a long one and presumably lying between coils of gut; but often a second examination later on will show it to be in quite a different posture. Then again there is apparent mobility when the appendix is adherent to the caecum and moves with the latter. In these circumstances it does not alter its position in relation to the caecum at later examinations.

Again it is impossible to be sure than an appendix does not fill and empty. The examinations may not coincide with a "full" period or it may be retro-caecal and not demonstrable when the caecum is full and who is to say that it has not filled and emptied whilst the caecum has obscured it? The normal appendix, therefore, may be seen full or may not be visible. If it is seen and is of normal size and contour, movable and not tender on pressure with no irregularities of the caecum about its junction, then I think we can reasonably assume it is normal. "Beading," that is when the opaque meal is broken up in the lumen of the appendix into segments, is not, I am sure, a sign of abnormality, but is due to a firmer consistency of the contents.

When the above conditions are present, but there is irregular narrrowing of the lumen at or near the base, then it is difficult to be certain whether a stricture is present unless there is dilatation and clubbing of the distal end or stasis therein. If there is absence of attacks of pain or tenderness on pressure in that region and no undue delay in filling and emptying of such an organ, then I would advise giving it the benefit of the doubt and leaving it alone. If, however, the evacuation of the contents is delayed after the caecum is empty, then removal seems indicated.

The Abnormal Appendix.

First, where the lumen is not obstructed, the opaque mixture fills the lumen, but the appendix may be drawn out and fixed either upwards, downwards or inwards or behind and even outside the caecum. It is tender on palpation and if the tip is fixed, the tautness of the organ may vary with the position of the patient. For instance in one patient the appendix which was very long with a broad lumen, passed vertically up behind the caecum and ascending colon, its tip being fixed firmly. In the upright posture it appeared very straight and taut, whilst in the horizontal posture the caecum ascended, relaxing the strain and all the tautness disappeared. In this instance the patient com-

[1] Read before the Radiological Section of the Victorian Branch of the British Medical Association on

plained of pain on stretching the arms above the head, when carrying weights *et cetera*, a symptom I have met with in other similar cases.

When the tip of the appendix is adherent towards the mid-line, attacks of increased frequency of micturition or dysuria occur and if it is adherent further back and lower down, rectal tenderness may be present, according as to whether the apex is adherent in the region of the bladder or rectum. In these cases the caecum may appear normal in shape and sharp in contour. Definite stasis of their contents is seen in such appendices, sometimes for a week or more. When the appendix fills and shows a definite constriction (by this I do not mean a temporary kink or bend or twist) which is always present and always tender on pressure with a dilated base or distal end or both, then I think, in the absence of other detectable cause for indefinite dyspeptic symptoms, it should be pronounced abnormal and removed.

If the appendix only partially fills, especially if only the proximal part fills and is irregular and tender and if its appearance is confirmed at a second examination, I think operation is advisable.

Such a condition was seen in a child of twelve years of age and the patient had recurrent attacks of acute colic across the upper part of the abdomen with slight rises of temperature, but with no definite tenderness at McBurney's point. Twenty hours after the meal a typical spasm of the transverse colon was present which had disappeared four hours later. The appendix was very stumpy and definitely tender on pressure. Dr. Moore removed it, confirming the observations and the boy was relieved of his attacks of colic.

If the lumen of the appendix is not seen to fill, only an indefinite opinion can be given, but in such cases the clinical symptoms and signs suggesting that the appendix is the offending organ can be strengthened by the following radiological signs, namely tenderness over the caecum and on its mesial side, delay of over twelve hours in the terminal ileum or in the *caput caeci* when the rest of the caecum and ascending colon are empty, with irregularity of the mesial side of the caecum about the caeco-appendiceal junction. Non-fiilling may be due to atresia of the appendix or fæcal stasis therein preventing the entrance of the opaque media. These signs, however, are not pathognomonic of an abnormal appendix, but are rather due to adhesions following inflammatory attacks and not infrequently seen after appendicectomy, especially when the operation has been done during an acute attack with abscess formation.

I mentioned before that I believe that chronic duodenal ulcer and abnormal appendix are not infrequently associated.

In one such case pressure on an adherent appendix caused pain radiating to the epigastrium. There was no doubt about the duodenal ulcer and the appendix proved to be partially retrocaecal and adherent at operation passing upwards and then outwards.

In quite a number of cases in which the appendix appeared definitely abnormal, pressure on it caused pain radiating to the epigastrium and in these cases the symptoms were often more suggestive of duodenal than of appendiceal pathology.

In some cases also I have seen a small gastric residue six hours after the meal, though no lesion could be detected in the stomach or duodenum suggesting a reflex pyloric spasm.

With regard to concretions I have seen clear areas in an otherwise filled appendix diagnosed as concretions. Now this is taking a lot for granted, as the area may be due to gas or to a patch of fæces not impregnated with barium. Recently I saw such areas develope in an appendix formerly evenly filled with opaque meal. I would diagnose a concretion only if it could be seen before an opaque meal or perhaps if a permanent clear area was present lightly outlined by a coating of barium. In my experience they can very rarely be diagnosed. May I be permitted to quote from an abstract appearing in the *American Medical Journal* of June, 1925, on page 679 of an article by R. H. Orndoff. He writes:

Tenderness of the appendix can probably be determined only in connexion with X ray observation and in rare cases it cannot be determined.

A tender appendix carries far more clinical value than tenderness at McBurney's point.

Also:

An appendix with a dilated distal end and a narrow lumen is pathologic.

Again:

Partial central constriction from angulation, fixation or other conditions of a pathologic anatomic character constitute a fairly large percentage of pathologic appendices which themselves do not warrant radical intervention, but when considered in the causation of the patient's general condition their removal constitutes one of the most successful operations.

And:

There is no abnormal position (of the appendix) without fixation.

A most excellent paper on "The Clinical Importance of Chronic Changes in the Appendix," by Franklin White, of Boston, Massachusetts, also appeared in the *American Journal of Roentgenology*, January, 1925, page 12, followed by a most instructive discussion and is well worth perusal.

I hope to show by these observations that valuable assistance can be given to the physician and surgeon by the radiologist in assisting them to come to a definite conclusion in such cases. I would like also to point out that in a number of cases in which the appendix lay in an unusual position, as shown by radiographs, the surgeon's task was simplified and the standard incision modified to meet the condition. In one case in particular there was an undescended caecum with the appendix up against the gall bladder. Without previous X ray examinations such cases can present great difficulties to the operating surgeon; they are much simplified when he knows the true position of the caecum.

I have not referred to pericaecal adhesions as evidence of prior attacks, as I wished to confine my observations as far as possible to the appendix itself and avoid extending the paper beyond a reasonable time, as I would like to demonstrate a number of films at the conclusion.

THE OCCURRENCE OF MOLYBDENUM IN THE EGG OF THE DOMESTIC FOWL: A PRELIMINARY COMMUNICATION.[1]

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THE increasing importance of the presence of heavy elements in tissues has led to the search for molybdenum. When X rays are "reflected" from the planes of a calcite crystal, a beam of homogeneous wave length is obtained, the wave length depending upon the angle between the incident beam of rays and the plane of atoms in the face of the crystal. A study of the effect of homogeneous X radiation upon the allantoic membrane of the fowl's egg reveals the presence of a reaction when the crystal is placed at an angle of 5° 49′. This wave length corresponds to the potassium absorption discontinuity of molybdenum. Consequently, it has appeared advisable to make an investigation for the presence of molybdenum in eggs. The materials examined have been the whites and yolks of fowls' eggs. The method adopted has been a modification of the one used by Dorothy Hall[(1)] and by J. Koppel.[(2)]

After the material has been ashed in a porcelain basin at a fairly low temperature, it has been transferred to a platinum basin. It had been found as the result of previous experiments that extraction of the ash with hydrochloric acid or *aqua regia* was not satisfactory. The ash has been treated with ammonium nitrate and then with a mixture of ammonium nitrate and sodium carbonate (1:2). After cooling, an aliquot portion of the mass has been dissolved in a minimum quantity of sulphuric acid and then made just alkaline with sodium hydroxide, the volume not being greater than about four or five cubic centimetres. From 0·3 gramme to 0·5 gramme of solid potassium xanthate has been dissolved in the solution and a few drops of 10% sulphuric have been added (the tube being agitated after the addition of each drop) till a purple colour has been developed which shows the presence of molybdenum.

Egg white has given a positive result to the test. The concentration of molybdenum from results so far obtained (a mean of results from eighteen eggs) is 0·01 milligramme per one hundred grammes of egg white, the colour obtained having the same intensity as that given by 0·01 milligramme of molybdenum as ammonium molybdate. A definite result for egg yolk has not yet been determined. Blanks have been tested by the use of ammonium nitrate and sodium carbonate in porcelain, silica and platinum vessels and the colour obtained has been no deeper than a faint pink (0·005 milligramme of molybdenum as ammonium molybdate gives a definite purplish tint). When 0·01 milligramme of molybdenum as ammonium molybdate has been evaporated in a platinum dish and ammonium nitrate added and later a mixture of ammonium nitrate *plus* sodium carbonate (1:2) a positive result was obtained, the recovery being quantitative on comparison with the standard.

The following elements gave no purple colouration with this test: Iron, strontium, magnesium, lead, potassium, chromium, cobalt, iodine, boron, uranium, nickel, arsenic, mercury, vanadium, bromine, chlorine and also phosphates, sulphates, nitrates.

Acknowledgement.

My thanks are due to Professor H. G. Chapman for suggesting this work and for his help and advice during its progress.

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(1) Dorothy Hall, *Journal of the American Chemical Society*, 1922, Volume XLIV, pages 1462 to 1465.
(2) J. Koppel: *Chemische Zeitung*, 1919, *Band* XLIII, *Seite* 778, 779.

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

Reviews.

THE NERVOUS SYSTEM IN MAN.

IN "Brain and Mind" Professor R. J. A. Berry has provided a concise and eminently readable account of the nervous system in man.[1] As the author points out in the preface, "it is only by proceeding from the simple to the complex that the physician, psychologist, psychiatrist and educationalist can hope to understand the phenomena of mind" and this principle underlies the whole scheme of the work. In fact the author has succeeded so well in guiding the reader from the simple to the complex that the anatomy of the central nervous system has largely been shorn of its difficulties. The diagrams and illustrations which are for the most part clear and free from unnecessary detail, are most helpful supplements to the text. In a work of this size we should have liked to see some references to the researches of Brouwer and others on the visual tracts and the cerebellum, especially as the author has given some consideration to the phylogenetic aspects of the nervous system.

From the sound foundation of the first part of this book we pass on to the more controversial matter contained in the second section, "The Nervous System in Health and Disease." When Professor Berry commits himself to such a statement as "the phenomena of mind can only be correctly interpreted in terms of neurones and neuronic arcs" (page 489), he at once defines his attitude and discloses the limitations of his survey of mental disorders. For throughout the book there is repeated the implication that underdevelopment of the supragranular cortex is one of the most important factors in mental disorders and cerebral amentia, as Professor Berry terms it, receives a corresponding amount of attention. For example he states: "It would certainly appear, on clinical grounds, that psychasthenia has much in common with cerebral amentia." While no one can dispute the maxim "no neurone, no mind," which Professor Berry has spared no pains to emphasize, the fact must not be overlooked that the most intensive study of the minute structure of the nervous system has not hitherto revealed the clue to behaviour. The nervous system provides the mechanism for selection and the final common path, as Sherrington has termed it, for impulses derived from a variety of sources and changes in the alimentary or the endocrine systems, for example, may have the most profound influence upon conduct. Professor Berry would therefore appear to have taken too narrow a view when he states:

[1] "Brain and Mind or the Nervous System of Man," by R. J. A. Berry, M.D., F.R.C.S., F.R.S. (Edinburgh); 1928. New York and Melbourne: The Macmillan Company. Royal 8vo., pp. 620, with illustrations. Price: $8.00 net.

"The form of the nervous system is always correlated with the behaviour of the animal possessing it. In other words its psychology or behaviour is entirely dependent upon its neurology."

In his plea for the better examination of the ament Professor Berry deserves every support. But here again, although he does not overlook the value of psychological tests of function, he would appear to stress overmuch the significance of structure, particularly of head measurement. From the practical standpoint it is of less interest to the psychiatrist to know that a head measurement below a certain figure in all probability indicates a subnormal depth of supragranular cortex than to apply tests of general intellectual capacity and performance which can show him what the individual can do. The author recommends as a minimum examination: Estimation of the cubic capacity of the brain, estimation of standing and sitting stature and the weight, estimation of the grip and vital capacity, the Binet and Porteus tests. Of these the psychological rather than the anthropometric tests indicate the ways in which a social misfit with a limited intellectual endowment can be helped to a better adjustment. While Professor Berry points out how cerebral aments may swell the ranks of the "criminals, thieves, prostitutes, ne'er-do-wells, antisocial cranks and quacks, and inefficients of all classes of life," he does not appear to regard the training of defectives as offering much in the way of prevention of antisocial attitudes or of treatment. As Foster Kennedy remarks in the introduction, "perhaps Professor Berry's emphasis on neuronic form as such tends to becloud somewhat his own demonstration that education is needed for the evolution of perfect neurones and discipline and good habit for the making of fine character." Even the ament may sometimes be taught to curb the demands of his infragranular cortex.

The author has little sympathy with the suggestionists and the psychoanalysts. "Baudouin," he states, "appears to ignore, or at least not to be familiar with the most elementary physiological evidence. He states that sleep is very ready to obey suggestion and that by autosuggestion we shall be able to go to sleep almost instantaneously, prescribe the exact hour of waking and prohibit nightmares." Professor Berry attempts to show the absurdity of such a claim in these words: "To prohibit nightmares without cutting off all incoming exteroceptive, proprioceptive and enteroceptive impulses, that is to kill the patient, is a physical impossibility. Nor is it possible to prevent nerve cells primarily charged with such impulses from functioning during sleep." Professor Berry has evidently been singularly unfortunate in his experience of psychotherapy. His remarks about the Freudian school are similarly unsympathetic and he would appear to have been guided more by other critics than by a first-hand acquaintance with psychoanalytic doctrine. With the reservation that there is an excessive bias towards a physiogenic explanation of mental disorders, this book is commended to the notice of all who have to deal with the mind in health or disease.

A TEXTBOOK ON SURGERY.

IN Ashhurst's "Surgery: Its Principles and Practice" the arrangement of the subjects is conducted upon more or less conventional lines.[1] That is to say the opening chapters deal with inflammation, tumours and surgical technique. These subjects are very well handled, especially perhaps the chapter on minor surgery wherein is included much useful information on blood transfusion, aspiration, phlebotomy and kindred matters.

The reader will turn eagerly to the subject of shock, hoping for a final elucidation of the nature and cause of this elusive condition; but he will find that it is still vague and shadowy and hung with the same old theories. Nevertheless the manner in which the subject is discussed is complete and sound.

Fractures and dislocations are dealt with in an efficient manner more or less on the usual lines.

The author writes on the whole subject of tuberculosis in a manner that convinces us of his wide experience. The way in which symptoms and signs arising from cranial injury is explained is extremely good. Seldom have we read such a clear, lucid and complete exposition of the subject. In the treatment of such, the possible danger of lumbar puncture is made evident. Incorporated in the section of injury to the spine are handy charts of reference concerning the level at which the injury has occurred. That the book is well up to date is shown by the inclusion of an account of "Lipiodol" injections for the location of intraspinal tumours.

There is a very good article on hernia and the different types are exhaustively and lucidly considered. Abdominal problems would appear to be a pet subject with the author as they are very fully and masterfully dealt with. Not much of the subject matter is open to criticism as the theme proceeds along sound and generally accepted lines.

The foregoing are merely some of the statements that can be made on reading through this work. Were they multiplied, their tenor would be the same. The author has not achieved anything startlingly original, but if his aim has been to present us with a picture of the principles and practice of modern surgery his effort has been eminently successful.

NEUROLOGICAL EXAMINATION.

THE fact that the manual on the clinical examination of the nervous system by Dr. Monrad-Krohn[1] has reached a fourth edition speaks for itself. We have already commented favourably on previous editions of the book and in regard to this edition may remark that it does not present much change. Indeed, as the author tells us in his preface, while the book has been submitted to general revision, the alterations are of minor character and the only noteworthy addition is a brief chapter on the interpretation of X ray pictures of the skull.

For those unacquainted with the book we may say that it is a product of the realization that the diagnosis of many neurological conditions demands a searching and systematic examination and such examination demands a knowledge of many methods. To memorize all these methods and the significance of what they may reveal is impossible for the ordinary individual. Therefore, to have at hand a manual such as this which is concise, correct and informative is an obvious advantage.

THE INTRODUCTION OF ETHER ANÆSTHESIA.

A PARTIAL perhaps, but apparently very faithful, account of the life of Crawford W. Long, the first user of ether for surgical anæsthesia, has been published by his daughter Frances Long Taylor.[2] The controversy as to whom the credit of this great discovery was due, raged furiously many years ago, the contestants being Wells, Morton and Jackson. These men certainly all played a great part in the introduction of the use of this anæsthetic, but on the whole the merit of priority seems indubitably the right of Crawford W. Long, a country doctor in Georgia, for he performed the first operation under its use on March 30, 1842, the operation being removal of a sebaceous cyst of the neck. His claims to the distinction were publicly advocated by Marion Sims so late as 1877, only a year before Long's death. No doubt the Civil War in the 'sixties made it impossible in America to advance these claims at a time closer to the actual event. However, better late than never and if this little book, written by his daughter, convinces the world of medicine of her father's contribution to the alleviation of suffering, it certainly will not have been written in vain. On the whole it is quite a convincing story.

[1] "Surgery: Its Principles and Practice for Students and Practitioners," by Astley Paston Cooper Ashhurst, A.B., M.D., F.A.C.S.; Third Edition, Thoroughly Revised; 1927. Philadelphia: Lea and Febiger. Royal 8vo., pp. 1170, with illustrations. Price: $10.00 net.

[1] "The Clinical Examination of the Nervous System," by G. H. Monrad-Krohn, M.D., F.R.C.P.; Fourth Edition; 1928. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 226, with illustrations. Price: 7s. 6d. net.

[2] "Crawford W. Long and the Discovery of Ether Anæsthesia," by Frances Long Taylor, with a foreword by Francis R. Packard, M.D.; 1928. New York: Paul B. Hoeber, Incorporated. Demy 8vo., pp. 250, with illustrations. Price: $4.00 net.

The Medical Journal of Australia

SATURDAY, JULY 21, 1928.

The School of Hygiene and Tropical Medicine.

IT has been announced that the Federal Public Works Committee has issued a report on the proposal to establish a school of hygiene and tropical medicine within the grounds of the University of Sydney. The committee has considered the scheme on which the proposals were based, that the school should be financed by the Commonwealth Government and controlled by a council having representation of both the Government and the University. The report has been entirely favourable and it may be assumed that the institution of the school has been sanctioned. It is stated that £30,000 will be set aside for the initial expenditure and that an annual sum of £9,000 will be provided for maintenance.

The history of the movement is a relatively long one. It will be remembered that in the early years of the present century Thomas Anderson Stuart planned the establishment of a school of tropical medicine at the University of Sydney, more particularly for the purpose of collecting information concerning the insect vectors of disease. He held the opinion that Sydney was favourably situated for such a purpose. The facilities for research of this kind were thought to be superior to those in existence in other parts of the Commonwealth. Port Jackson possessed large shipping connexions with other parts of the world and especially was the first port of call of ships sailing from the islands of the Pacific. Anderson Stuart maintained furthermore that Sydney was ideally placed for the purpose of sending expeditions to the tropical areas of Australia, to New Guinea and to the islands in the Pacific. The project became actual about the year 1906 when a strong committee was formed as a result of several conversations between Anderson Stuart and other prominent men of the day. The members of the committee included the late Harry Allen, the late Edward Charles Stirling, the late Anderson Stuart, Dr. W. W. Love, Mr. Atlee Hunt, Dr. W. O. Norris and Dr. J. S. C. Elkington. The Sydney scheme had been opposed by Bishop Frodsham and had been replaced by another. This was to establish an institute of tropical medicine at Townsville. There is little doubt in the minds of those who are now able to judge the matter impartially and in the light of subsequent developments, that Anderson Stuart's original scheme was the better and that had it then been established, much progress would have been made by this time.

Closely connected with the same subject was the proposal made several years ago and strongly supported by this journal for the institution of a Pacific medical service. The claim was again made for Sydney that on account of its geographical position and of its other facilities, this city should be made the headquarters of the medical service. There were certain international obstacles to the scheme and for a time no progress was registered. The next stage owes its initiation to the Royal Commission on Health. The Commissioners devoted a section of their report to the subject of chairs of preventive medicine and tropical hygiene. They had taken evidence on a proposal to establish schools and chairs in these subjects and concurred with the proposal. In view of the existing conditions in Australia, the Commissioners advocated the establishment of one school at the University of Sydney with a chair of preventive medicine. A chair of tropical hygiene could be instituted at a later date. The Commissioners expressed the opinion that records of the medical administration of the Pacific Islands should be collected and condensed with a view to their distribution. It will be noted that the conception of the Royal Commissioners was not to expand the existing plan of teaching and training for the diploma of public health, but to develope the modern conception of preventive medicine, with its basic principle of attack on the source of infection in the infected individual. It may be mentioned that the facilities provided by the Commonwealth Department of Health for the study of tropical medicine and hygiene at Townsville as part of the course for the diploma of tropical medicine have attracted but few graduates.

The last stage in the evolution of the scheme is the definite proposal made by the Commonwealth Government to the Senate of the University of Sydney to establish the school of hygiene and tropical medicine. There were obstacles to be overcome and objections to be met. While the bodies concerned were in substantial agreement, it was thought that the views of individuals should be ascertained before the project was translated into action. Evidence was taken by the Public Works Committee and the opposition to the scheme was examined and evaluated. It now appears that after careful consideration of all the evidence the committee has decided that the establishment of the school will be to the advantage of the Commonwealth of Australia and of benefit to the races of the Pacific.

A quarter of a century has elapsed since the proposal was first broached. Much water has flown under the bridge and there have been many changes since the start. It is probable that the objects in view when Anderson Stuart conceived the idea, were less broad and less clearly developed than were the ideas of the Royal Commissioners. It is essential that a modern school of hygiene and tropical medicine must be conceived in a spirit of prevention. Those working at the school must investigate the ætiology of disease and its pathogenesis and must gather knowledge concerning the best methods of keeping Australia, including its tropical regions, and the adjacent lands healthy and of making them healthier. It is easy to teach when knowledge has been acquired and satisfactory training is therefore inseparable from original research and organized study. The establishment of the school will be another monument to the wisdom of the Royal Commission on Health.

Current Comment.

TUMOURS OF THE NERVUS ACUSTICUS.

In the recent discussion by the South Australian Branch of the British Medical Association on tumours of the cerebello-pontine angle, tumours of the *nervus acusticus* were specially considered. The pathology of the latter growths has been the subject of much discussion. In his paper which appeared in the issue of June 30, 1928, de Crespigny states that the tumour is a true nerve tumour—"neurofibroma or neuroglioma." Here lies the difficulty. If these tumours are neurofibromata, they arise from connective tissue and are of mesodermal origin; if they are neurogliomata, they must be regarded as ectodermal. Bland Sutton considers tumours of the *nervus acusticus* under the same heading as plexiform neuroma which he describes as being essentially a fibromatosis. Ewing discusses these tumours under the heading "Acoustic Neurofibroma" and states that the structure is usually that of a simple neurofibroma, but that some very cellular growths approach the type of neurosarcoma. He points out that glial tissue has several times been demonstrated and that from this element a sarcomatous process may be derived.

The histology of tumours of the *nervus acusticus* has recently been discussed by C. P. Rhoads and W. P. Van Wagenen.[1] They state that those who regard the tumours as arising from nervous tissue have drawn their deductions from three lines of reasoning. The first is that the microscopical appearance of the tumours is not typical of that seen in ordinary fibromata. The second has been advanced by French investigators. Since the fibres became sharply coloured with Victoria blue and since this dye stains neuroglial fibrils well, the fibrils were regarded as being neuroglial. It has been found, however, that Victoria blue stains fibroglia, fibrils and fibrin as well as it does fibrils of neuroglia. The third argument is the most important; it is based on the embryological work of Harrison and of Harvey and Burr. Harrison showed that when the neural crest is removed from frog embryos of a certain age, peripheral nerves develope without a sheath of Schwann. Harvey showed that the brain of an amblyostoma embryo, transplanted without the neural crest into a second embryo, developed without the formation of a pia-arachnoid. When the neural crest was transplanted with the brain, the leptomeninges developed normally. There is thus strong evidence that the sheath of Schwann and the pia-arachnoid are ectodermal in origin. If this evidence were accepted and if it were true as Verocay suggested that tumours of the *nervus acusticus* arise from the sheath of Schwann, the tumours would necessarily be regarded as ectodermal in origin. Rhoads and Van Wagenen hold that this line of reasoning is open to attack on three grounds. In the first place Weed has demonstrated that the pia-arachnoid forms in transplanted embryo brains only when chorioid plexus as well as neural crest is included in the transplant. In this way the failure of Harvey and Burr to grow meninges on a brain transplanted without neural crest may have been due to an absence of chorioid plexus. In the second place many observers claim that tumours of the *nervus acusticus* arise from the perineurium and not from the sheath of Schwann; the former is regarded by most anatomists as being fibroblastic in nature. A third explanation is that the neural

[1] *The American Journal of Pathology*, March, 1928.

crest may contain undifferentiated mesenchymal cells which might give rise to the nerve coverings. Moreover, there is a group of anatomists who maintain that connective tissue may develope from ectoderm as well as from mesoderm.

Rhoads and Van Wagenen state that there are five criteria that a given tissue is derived from fibroblast. These are the morphology of the cell, the presence of collagen, the presence of elastic fibrils, the presence of fibroglia fibrils and the presence of reticulum. They then describe their findings on the examination of fifty-four specimens obtained from Cushing's clinic and claim that all these five points were exemplified in the tumours.

From this discussion it is seen that the balance of evidence is in favour of a mesodermal origin of these tumours. Unfortunately there is as yet no agreement as to the origin of the various components of a nerve. Until this is settled there can be no definite conclusion in the matter of nerve tumours. Rhoads and Van Wagenen make no reference to tumours which contain glial tissue, such as mentioned by Ewing. Glial tissue is generally regarded as being of ectodermal origin and the presence of glial cells in a tumour of the *nervus acusticus* would be difficult to explain on a purely fibroblastic basis. As a matter of fact Rhoads and Van Wagenen describe some isolated fibroblasts in which a nucleolus was present and point out that it would be quite easy to regard these as nerve cells. Embryological research probably holds the key to the full explanation.

LYMPHATICOSTOMY IN ACUTE PERITONITIS.

IN November, 1922, Costain reported some experiments which he had carried out on dogs. He produced septic peritonitis by the ligation of the appendix and found that if the thoracic duct was opened in the neck of the animal, recovery took place. All the control animals died. In the following year he reported that a nine year old child suffering from pneumococcal peritonitis had recovered quickly after the thoracic duct was opened in the neck. He undertook this work on the assumption that death is due to the absorption of toxic materials through the lymphatics. The results of this form of treatment have been variable. In order to determine the value of lymphaticostomy K. Paterson Brown has recently carried out a series of experiments on rabbits, cats and dogs.[1] He used a 2% solution of colloidal silver in saline solution. When the silver solution was injected into the peritoneal cavity of rabbits, it appeared very quickly in the thoracic duct. With cats and dogs the appearance of the silver in the thoracic duct was much delayed; no staining took place until two hours had elapsed and then only faint traces could be seen at the lower end of the duct. The diaphragmatic lymphatics drained to the internal mammary lymphatics which were well stained after half an hour of injection. In a dog which died of acute peritonitis, the glands draining the internal mammary vessels were enlarged and congested, but the thoracic duct was normal. It was also found that if the rabbits were kept in an upright position, a definite delay occurred in the appearance of the silver in the thoracic duct. Brown concludes that the lymphatic path of rabbits is different from that of cats and dogs. His work certainly indicates that this is so, but at the same time the fact that the experiments were all carried out under anæsthesia excludes to a large extent, if not entirely, the factor of intraabdominal pressure, the importance of which has been emphasized by Florey (see THE MEDICAL JOURNAL OF AUSTRALIA, May 19, 1928). His observations justify his conclusion that lymphaticostomy cannot be recommended for patients with acute peritonitis; it must also be remembered that by draining the thoracic duct these persons will be deprived of much of the chyle which is so necessary to their sustenance.

[1] *The British Journal of Surgery*, April, 1928.

THE REPAIR OF NERVES AND TENDONS OF THE HAND.

THE rôle of excessive fibrous tissue in the production of deformity is well exemplified in the case of injuries to the hand. In extensive injuries cicatricial tissue around tendons, nerves, lymphatics and blood vessels not only produces mechanical disability, but trophic disorders the extent of which is sometimes out of proportion to the original violence.

In attempting to reconstruct a damaged hand it is necessary to bear these facts in mind and to plan the operative procedures accordingly. The stiff joint should be mobilized before attempting to graft its controlling tendon and similarly it would be useless to place a free tendon graft where the nerve supply had been destroyed until the nerve had time to regenerate. Sterling Bunnell, of San Francisco, has given this subject an intensive study.[1] His results show that much can be done to restore function, if attention is paid to the detailed physiology and pathology. He lays down fundamental principles of treatment, many of which are not new, but are of such general application that they will bear repetition.

Cicatricial tissue should be excised widely until healthy tissue is encountered. Where skin grafting is necessary, tubed pedicled grafts are used. Free tendon grafts may be used and provided elastic paratenon or other gliding mechanism is carefully placed they will free themselves from adhesions and function well. Where possible nerves should be sutured and, if necessary, joints are flexed to relieve tension. Where it is impossible to bridge the gap by simple suture, grafts from the sural nerves regenerate almost as quickly as after ordinary suture. Joints should be mobilized by mild continuous force and all trauma should be reduced to a minimum. Ischæmia should be as short as possible and asepsis should be rigid.

[1] *The Journal of Bone and Joint Surgery*, January, 1928.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Puncture of Lymphatic Glands for Purposes of Examination.

C. E. FORKNER (*Archives of Internal Medicine*, November 15, 1927) has reported the study of material obtained by the puncture of the lymphatic glands of thirty patients suffering from disease of these structures. In the majority of instances the tissues of the gland were submitted to pathological examination. Lymphatic glands obtained from cases in which there was no apparent pathological process of the lymphatic glands, were used as controls. The conditions studied included different types of lymphoblastoma, tuberculosis, benign hyperplasia and carcinoma. It was found that a distinctive cytological picture was obtained in all instances. Thus, in benign hyperplasia there was an increased number of polymorphonuclear neutrophile cells. In tuberculosis epithelioid cells were abundant and giant cells were found. In carcinoma the cells were almost entirely of one type and no lymphoid cells or other normal constituents of lymphatic glands were found. In chronic lymphatic leuchæmia, with or without changes in the blood characteristic of leuchæmia, cells of the lymphatic series dominated the picture. In Hodgkin's disease large pale endothelioid cells were present in considerable numbers, giant cells were seen and eosinophile cells were often abundant. In rapidly progressing lymphoblastoma, other than leuchæmia and Hodgkin's disease, there was a definite increase in the reticular cells and all types of typical and atypical lymphoblasts were seen. The author points out that the observations are few and do not permit of unequivocal statements. He states, however, that the method makes it possible to obtain information concerning the cytology, bacteriology and pathology of disease processes. By this method it is possible to follow the spontaneous morphological changes in disease and those induced by treatment. The character of the cells obtained assists also in the formulation of prognosis.

The Staining of Gram-positive Organisms in the Tissues.

R. D. LILLIE (*Archives of Pathology*, May, 1928) has given details of a quick method for staining Gram-positive organisms in the tissues. Paraffin sections of about five microns thickness or frozen sections fixed to the slide by the celloidin method, are brought down to water in the usual way. The sections are stained for thirty seconds with ammonium oxalate crystal violet. For tubercle or lepra bacilli staining for a longer period or heating is often necessary. Heating for ninety seconds on a hot plate at from 50° to 52° C. has been found satisfactory. The sections are then washed in tap water. Lugol's solution is applied for thirty seconds. The sections are washed in tap water. Acetone is applied until no more colour is removed, this takes about ten to fifteen seconds. The sections are washed in water and they should not be allowed to dry after the acetone is applied. They are counterstained with 0·5% safranin in water for thirty seconds. They are washed in water. They are dehydrated and differentiated with acetone; a certain amount of red comes out in this process, leaving the cell nuclei deep red and the cytoplasm pink. They are cleared with xylol. This method can be carried out in three minutes and is claimed to give clear-cut results. Gram-positive bacteria are blue black; Gram-negative organisms, red; cell nuclei, deep red; cytoplasm of lymphocytes and plasma cells, moderately deep pink; cytoplasm of other cells, pale pink; fibrin, pale pink; collagen, usually pink, sometimes light violet in dense bundles.

Cretinism.

ELIZABETH C. EAVES AND MARGARET M. CROLL (*The Journal of Pathology and Bacteriology*, April, 1928) have reported a case of nervous cretinism in which a histological examination of the organs was made. The patient was a girl who died at the age of fifteen years from bronchopneumonia caused by food entering the trachea. The history of the child was characterized by the three stages described by Horsley in 1886 in his experimental work on removal of the thyreoid from monkeys: A stage of tremor and convulsions, a mucinoid stage in which imbecility occurs and motor symptoms cease, a cachectic stage in which mucin is lost and marasmus occurs. At *post mortem* examination a piece of tissue was found in the usual position of the thyreoid; it consisted of two lobes and a small isthmus and weighed a little over two grammes. On microscopical examination no trace of thyreoid tissue was discovered. No parathyreoids were found. The suprarenals were large in proportion to the body weight and the relative proportions of cortex and medulla appeared normal. The pancreas appeared to be rather small, but was probably about normal in proportion to the body weight; the same was found in regard to the heart, liver and kidneys. The amount of islet tissue appeared to be greater than normal and the authors measured the percentage amount of it in different parts of the organ by a method somewhat similar to that used by Heiberg. The mean figure was 3·54% and this is higher than the figure given by the majority of observers for the adult human pancreas. The pituitary weighed 0·24 grammes; this is within the normal range for the body weight and too small for the age. The posterior lobe was relatively much smaller than it should have been. In the anterior lobe the cells everywhere were chiefly chromophobe and confluent and considerably vacuolated. In some situations there was a considerable amount of colloid between the cells or in the sinuses. Occasionally the cells were arranged around the colloid in vesicles slightly like those of the thyreoid gland. The authors refer to the generally accepted view that absence of any one ductless gland may bring about alterations in the other ductless glands and state that their findings lend support to the view that there is a relationship between the thyreoid, the pancreas and the pituitary. As far as they can ascertain the increased islet tissue in a human cretin has not been observed before. Lorand found that removal of the thyreoid in animals leads to an increase in the numbers and size of the islets. The failure of other observers to see any change in the pancreas of animals whose thyreoids have been removed, may have been due to a question of age and the time which elapsed after thyreoidectomy. There is a difference between the chromophobe cells in hypopituitarism and those in the cretin's pituitary; in the former the cells are discrete and usually small, in the latter they are confluent. The authors suggest that former overactivity has been followed by exhaustion. There may be an association between the large size of the islets of Langerhans and the condition of the pituitary. One of the authors has shown that repeated injections of insulin in rabbits bring about increased activity of the cells of the *pars anterior* and *intermedia*. Further experiments which are not yet completed, indicate that increase in the amount of insulin exhausts the gland.

Calcium Compounds and the Chicken Tumour Virus.

HAVING discovered, while investigating the adsorption of certain viruses by means of particulate matter, that precipitated chalk inactivated the chicken tumour virus, Margaret R. Lewis and Howard B. Andervont (*Bulletin of the Johns Hopkins Hospital*, April, 1928) have investigated the effect of a few of the calcium compounds on the same virus. They found that the addition of calcium oxide, of calcium monobasic phosphate and calcium hexose phosphate in quantities of 0·5%, 0·75% and 1% to chicken tumour extract, inactivated the virus and prevented the growth of a tumour. The virus was adsorbed and fixed when 25% of precipitated calcium carbonate was added to tumour extract. When small amounts of chalk (2%, 1%, 0·5%) were added to the tumour extract, the growth of a tumour was sometimes prevented. The addition of 2% of plaster of Paris to the tumour extract inactivated the virus, but when only 1% of plaster of Paris was added to the extract, both the supernatant fluid and the washed deposit caused the growth of a tumour when injected into the breast of a chicken. The authors admit a probability that the inactivation of the tumour virus may in some instances have been due to a chemical change rather than its adsorption by the particulate matter. They claim, however, that when the

calcium compound took up so much of the virus that the deposit, although it was well washed, was able to bring about the growth of a tumour, the phenomenon might be interpreted as corresponding to the adsorption but not fixation of the virus by the calcium particles. Another fact pointing to adsorption is that although calcium carbonate always inactivated the virus when added to the tumour extract in large amounts, when small amounts were added it frequently failed to do so and it became possible for the washed deposit from even as little as 0·4% of calcium carbonate to bring about the growth of a tumour.

The Examination of Body Fluids for Tumour Cells.

A. P. ZEMANSKY (*The American Journal of the Medical Sciences*, April, 1928) has made an analysis of the results obtained by the examination of one hundred and thirteen samples of fluid, mostly pleural and peritoneal, for tumour cells. The results were checked by the subsequent examination of tissue. After the fluid was centrifuged the supernatant layer was poured off and a 10% solution of formalin was added. The deposit was hardened in this way and it was then cut into halves from above downwards. These halves were embedded in paraffin, cut, stained and examined. The findings which were most valuable in the diagnosis of the presence of tumour, were: (i) fragments of tissue with definite arrangement of cells and stroma, such as glandular acini, (ii) multiple groups of large, deep-staining cells, giving the slide a mottled appearance, (iii) finer cellular changes such as extreme irregularity of cell outline, eccentricity of nucleus, extremely large size of nucleus, multinucleation and typical and atypical mitotic figures. The number of cases in which a diagnosis of the presence of tumour was made was 39; this finding was verified in 34 instances and disproved in four. The number of cases in which it was reported that no tumour was present, was 74; this was verified in 31 instances and disproved in 43. The report of the presence of tumour is thus claimed to carry a high percentage of accuracy, while the report of absence of tumour is of little value. In this respect the examination of fluids does not differ from any other laboratory procedure. In discussing the five cases in which a report of the presence of tumour was disproved by subsequent examination, the author states that the mistakes should not have been made and that by the application of criteria, previously mentioned, such errors can be avoided. At the same time he believes that a few instances will always occur in which the most experienced examiner will find it almost impossible to make a decision. It is to be expected that cells or fragments of tissue will be found in a little more than half of the effusions from malignant tumours. The type of tumour can be determined from the character of the cells in the effusion in about one-third of the cases. There is about twice the likelihood of detecting a carcinoma from examination of an effusion as there is of detecting a sarcoma. The examination of sputum, gastric contents and urine for tumour cells is of little value.

Primary Double Cancer.

B. M. FRIED (*Archives of Pathology*, April, 1928) states that primary double or multiple cancer is a malignant condition in which a person is affected by two or more cancers which have started spontaneously and independently either in different organs or in different structures in the same organ. According to Billroth two cancers found in the same organism may be considered as being independent growths only (i) when they vary in structure, (ii) when each tumour can be traced to its respective matrix and (iii) when metastases can be traced to the respective mother tumour. These requirements can rarely be fulfilled. The author has reported the history of three patients who suffered from primary double cancer. The first was a man aged seventy years. At *post mortem* examination two tumours were found, a primary carcinoma of the tail and body of the pancreas with metastases to the lungs and the liver and a primary carcinoma of the prostate. It was concluded that the pancreatic tumour arose from the epithelial cells forming the acini. The metastases in the lungs and the liver closely resembled the pancreatic growth. In the prostate no pathological changes could be seen with the naked eye, but histological examination revealed a cancer in the early stages of development. It was a cancer of multicentric origin, in other words it was a growth which started at different points in the organ. It affected the acinar epithelium and the glandular units were often completely obliterated by malignant cells. The second patient was a woman, aged fifty-one years. She suffered from an epidermoid cancer of the *cervix uteri* and a colloid carcinoma of the anus. The third patient was a woman, aged thirty years. She suffered from a scirrhous carcinoma of the right breast and a colloid adeno-carcinoma of the left breast. The author points out that there are two possible explanations for the rare occurrence of primary double cancer. According to the first of these the rarity of such tumours may be attributed to the state of immunity or resistance to cancer. Jensen showed that in certain races of mice there was a natural immunity towards cancer and Ehrlich produced evidence to show that a state of resistance to malignant tumours could be induced experimentally in rats and mice. Although the intimate mechanism of natural or acquired resistance to cancer has not as yet been elucidated, the facts established by Ehrlich have been corroborated by numerous recent observers. If the view is accepted that man and animals display the same biological peculiarities towards epithelial malignant disease, a person with an actively growing cancer ought to resist the development of a new one. The author is of the opinion that it would be premature to attribute the rarity of primary double cancer to immunological tendencies and he refers to findings by numerous workers which indicate that the results of experiments are often contradictory and that it would be misleading to draw analogies between animals and man. The second possible cause for the rarity of primary double cancer may be due to the fact that a person with cancer somewhere in the body succumbs to the disease before a second epithelial malignant tumour has had time to develope. He regards the second possibility as the dominating factor.

Paget's Disease of the Glans Penis.

M. P. SUSMAN (*The British Journal of Surgery*, April, 1928) reports a case of Paget's disease of the *glans penis*. Only thirty-four extramammary cases of Paget's disease have been reported. The external genital region was affected in fourteen of these and the *glans penis* was the site in seven. The patient was a painter aged sixty years. He complained of an itchy excoriation around the urethral meatus of a year's duration. Evidence of visceral syphilis was found on examination. The penis was amputated just in front of the pubes. The microscopical appearances are similar to those seen in Paget's disease of the breast. The normal epithelium is seen to pass abruptly into a thickened patch; the thickness is due to proliferation of the Malpighian layer and it extends into the corium. There is a general round cell infiltration. Under high power the characteristic Paget cells are seen; these are round bodies, larger than normal epidermal cells, with homogeneous cytoplasm and deeply staining nuclei. Several undergoing mitosis were found. The author lays stress on the general similarity between the histological appearances of this lesion and those of basal-cell carcinoma (rodent ulcer).

Tuberculous Aneurysm of the Hepatic Artery.

W. P. THOMPSON (*Bulletin of the Johns Hopkins Hospital*, March, 1928) points out that, although aneurysm of the hepatic artery has frequently been reported, in no instance has tuberculosis been regarded as either an ætiological or a complicating factor. He reports a case in which he holds that tuberculosis can quite definitely be regarded as the cause of this condition. The patient was a man, aged thirty-four years, who was first seen in March, 1917, on account of small suprapubic sinus. This was found to be tuberculous. After it had been excised twice, it remained closed and the patient was without symptoms for eight years. He then complained of pain in the upper part of the abdomen. He became jaundiced and passed blood in his stools. He had a persistently high temperature and was regarded as suffering from empyema of the gall bladder with acute cholangitis. Later on the gall bladder was opened and drained. The

gall bladder was œdematous and contained blood clots and blood-stained bile. The patient died eight days later in coma. At autopsy an ulcer was found in the duodenum. The intestinal tract was filled throughout its course with blood in varying stages of digestion. The bile ducts were greatly enlarged. In the common hepatic duct there was a projecting thrombus-like mass; it surrounded an elongated tear which had a ragged recurved margin. The hole communicated with an aneurysmal sac and this opened directly into a branch of the hepatic artery. Hyaline material was found in the arterial wall at the point of rupture. The tissue surrounding the aneurysmal sac was almost entirely tuberculous with many scattered and epithelioid cells and occasional giant cells resting in a stroma of strands of connective tissue. In an adjacent lymphatic gland were found many fresh tubercles in which acid-fast bacilli were seen. Tubercles were scattered throughout the liver; in some of these early caseation was found.

Examination of the Blood.

H. B. NEWMAN AND F. M. HARVEY (*The Journal of Tropical Medicine and Hygiene*, April 2, 1928) state that with present-day methods of counting the red cells in the blood it is impossible to arrive at the exact total of the red cell content. They agree with Osgood that duplicate counts of the cells of any one patient made by the same observer should not vary to an amount exceeding 100,000 cells per cubic millimetre. Among the commoner errors made in using the hæmocytometer is carelessness in drawing up the blood to the exact mark on the pipette and another error is made by overstepping the upper mark in drawing up the diluting fluid. They suggest that by the use of a considerably longer piece of rubber than is usually provided with the pipette it is possible to obtain a better view of the marks on the pipette and that errors are less likely to occur. Further, the aspiration of fluids into the pipette can be more accurately controlled if the end of the rubber tubing where suction is applied, be throttled. This is effected by applying suction through a hypodermic needle; the point of the needle is ground away and the needle is inserted into the lumen of the rubber tube until the rubber grips the front part of the base of the needle. The cover slip should not be too thin, otherwise the layer of fluid covering the scale may be of irregular depths. The authors have experimented with pipettes bought in the open market and they found great variation in the results obtained with them. They hold that only accurately calibrated pipettes should be used. In their opinion the most satisfactory is a pipette of the Piney type. An estimate based on the enumeration of the cells met with over eighty squares is unreliable and at least two hundred and fifty-six squares should be counted. More accurate results were made with the old Thoma-Zeiss slide than with the latest Leitz pattern. They also conclude that it is preferable to use a dilution of one in one hundred than one of one in two hundred.

Sympathico-Blastoma of the Adrenal.

MARTHA WOLLSTEIN (*Surgery, Gynecology and Obstetrics*, June, 1928) describes a series of nine adrenal sympathico-blastomata submitted to microscopical study either at operation or at autopsy. Three of these tumours occurred in boys and six in girls. The tumour involved the left adrenal in four patients, the right in four and both adrenals in one. The age of the children varied from three weeks to almost three years. It was found that they fell into five different types according to the location of the primary tumour and of its metastases. In the simplest type of case a small nodule was found in the left adrenal body, the greater portion of the organ was apparently normal and no metastases were present. In this tumour the typical structure of embryonal sympathetic cells and fibrils was present. The second type was represented by two cases. In both these the tumour was unilateral and attained a large size; metastases were present in the liver alone. The third type conforms to what has been known as the Hutchison type of round-celled sarcoma of the adrenal body. It was represented by two cases. In both these extensive metastases were present. In the fourth group there were two patients. In one both adrenals were affected and in the other one. In both instances an inoperable tumour was found lying in front of the vertebral column between the adrenals and the kidneys. The prevertible mass in each represented a second tumour focus. The final group comprised two tumours. Both were removed by operation and the younger child is alive one year and five months later. In these tumours two distinct portions were found on section. The firmer portion contained many large adult nerve cells with a distinct nuclei and nucleoli, while the other portion of the tumour was composed almost entirely of the embryonal type of sympathetic nerve cells with numerous fibrils representing axis cylinders. Chromaffin cells were not present and both tumours were classified as neuroblastoma and ganglioneuroma. The author also makes reference to the opinions of several authors on this condition and points out that on the basis of the most recent histological investigation Bailey and Cushing prefer the name sympathico-blastoma, because not all the cells are potentially neuroblasts since some of them may differentiate into chromaffin cells.

The Adrenal Glands in Pregnancy.

J. C. DONALDSON (*Anatomical Record*, April 25, 1928) concludes that during the period of pregnancy no change takes place in the relative volumes of the cortex and the medulla in the adrenal of the albino rat.

MORPHOLOGY.

Reversal of the Anterior End of the Spinal Cord.

S. R. DETWILER (*The Journal of Comparative Neurology*, April 15, 1928) gives an account of further work on the transposition of segments of the spinal cord and brain stem in amphibian embryos. In an earlier contribution he showed that when segments of the cord are reversed end to end, they tend to fit into the general gradient of proliferation, so that essentially a typical cord is developed. He also claimed that these experiments pointed to the view that, even though there be a certain inherent potential for growth and differentiation, the eventual size and cellular content of various cord segments are conditioned by their position with respect to the cord as a whole. In experiments, however, in which the anterior end of the cord (first three segments) are shifted caudally, he obtained results which show that the anterior end of the cord possesses an inherent capacity for extensive proliferation as compared with the caudal end of the medulla and with segments of the cord lying caudal to the unit involved in the experiments. This position of the cord is not greatly affected by position such as has been observed in experiments involving more caudally lying spinal segments and its extensive proliferation capacity tends to maintain itself even in regions where proliferation is normally much lower.

Loss of Purkinje Cells with Age.

TETSUO INUKAI (*Journal of Comparative Neurology*, April 15, 1928) gives an account of his researches into the number of Purkinje cells in the cerebellum of the albino rat. He finds that with advancing age the number of Purkinje cells decreases regularly in all parts of the cortex and in both sexes. The loss of motor control in the aged albino rat is similar to that shown by the aged man, but in both instances the loss of the Purkinje cells is probably only one of several factors contributing to the deficiency observed.

Degeneration of Peripheral Nerve.

P. C. BUCY (*Journal of Comparative Neurology*, April 25, 1928) gives an account of his researches into the changes which take place in the complete neuron after section of its axon. First he studied the changes within the peripheral nerve and within the cell body together, in order that the two might be more closely correlated and secondly, he studied several nerves, both cranial and spinal, in order that he might compare the changes in each under the same conditions. The animal used was the rabbit. He finds that section of the motor division of the sciatic nerve in rabbits is not followed by axonal chromatolysis in the neurocyte. Following section of a nerve, union occurs in the absence of suturing or undue

tension in fourteen to twenty-one days. The earliest degenerative changes in the nerve were seen on the second day after sectioning. The earliest changes in the neurocyte were seen in the facial nucleus at the second day; others on the fourth day after section. The height of chromatolysis in all nerves was observed about the fourteenth to the twenty-first day and the height of chromolytic change occurred simultaneously with the union of the severed nerve. The cells of the facial and hypoglossal nuclei rapidly returned to normal by the fifty-sixth day, while the cells of the posterior root ganglia did not do so until one hundred days after section.

The Valves in the Human Thoracic Duct.

OTTO F. KAMPMEIER (*The Anatomical Record*, April 25, 1928) makes the following deductions from observations recorded in a paper on the numerical variability, position, function and fate of the valves in the human thoracic duct. Valves are laid down in the thoracic duct potentially in great superfluity. The extreme numerical variability of valves exhibited by it later is the result partly of the variable number initially designed, but to a greater degree the result of reduction in number of the original quota. Many valves never progress beyond the earliest tangible genetic phase of the *Anlage* and vanish soon after they have become manifest, but some advance far towards completion. The position of the surviving valves in the thoracic duct strongly illumines the rôle played by them in the lymphatic organization. Functional valves are found in that zone of the duct interposed between the aorta and œsophagus, that is, where these tubes cross one another diagonally to take up their position in the midplane, one above the other. One or more efficient valves were also found in the neighbourhood of the diaphragm. These observations would indicate that the valves are retained in regions which will suffer considerable direct pressure.

Many-boned Human Skull.

PHILIP B. ARMSTRONG (*Anatomical Record*, April 25, 1928) describes an adult human skull which has definite developmental abnormalities. These probably arose during the fœtal period and persisted into adult life. Several different types of abnormalities are described and of these two are regarded as important. First, the total number of bones making up the skull is increased to one hundred and sixteen. This increase in number is brought about by persistence of certain of the early developmental centres of ossification, by the occurrence of new or extra centres of ossification and by an excessively large number of sutural bones. Secondly, parts of some bones are partially or completely suppressed. This is most striking in the bones of the orbital roofs and floors and in the base of the skull. Other less striking abnormalities are seen in dentition, density of bone and sutural formation. All these abnormalities are attributed to the action of some condition which prevented deposition of the inorganic bone constituents and which was active through fœtal development and into the postnatal life of the individual. A mild persistent hyperparathyreoidism might possibly seem adequate to explain the maldevelopments found in this skull.

The Thymic Corpuscles of Hassall.

B. F. KINGSBURY (*The Anatomical Record*, April 25, 1928) supports the origin of thymic corpuscles from the cyto-reticulum following the view of Hammar and Dearth as opposed to that put forward recently by Jordan and Horsley who attempted to revive the old interpretation that Hassall's corpuscles are endothelial hypertrophies of obliterated capillaries or precapillary arterioles. The author interprets thymic corpuscles as expressions of growth transformation in an epithelium as modified and determined through loss of surface relations and under conditions of definite reticulation. He states that the comparison of thymic corpuscles and other epithelial growths in confined space, such as epithelial pearls, is justified and that the interpretation here presented requires no assumption of an adaptive significance underlying thymic corpuscle formation.

Motility of Leucocytes.

MARJORIE HENDERSON (*The Anatomical Record*, April 25, 1928) describes observations made on slide preparations of human blood set up in a box containing a thermostat so that the slides could be observed at any desired temperature for any period of time. Neutrophile cells seem to show an affinity for certain erythrocytes and appear to be capable of leaving a perceptible injury on the cells with which they come in contact. An endeavour was made to establish a standard of motility for normal neutrophile cells. This was found to be 36·66 μ per minute. In general, neutrophile cells showed a wide range in their velocity, one moving 53·9 μ and another 30·8 μ per minute. Neutrophile cells develope vacuoles and become non-motile after some hours at 37·5° C., while lymphocytes do not become vacuolated, but increase in motility. Observations on the response of leucocytes to increase in temperature, sodium citrate, weak alkali, weak acids and adrenalin are also recorded. The author gives a review of the literature and a lengthy bibliography.

Innervation of Skeletal Muscle of the Cat.

JOSEPH C. HINSEY (*Journal of Comparative Neurology*, October 15, 1927) gives an account of observations made on the innervation of voluntary muscle. The three nerve components to skeletal muscle, ventral root, dorsal root and sympathetic, have been isolated by degeneration. Each was studied in pyridin-silver preparations of the degenerated muscles. The following is a summary of his findings. In regard to the ventral root component of the *quadriceps* the motor plates were supplied by myelinated fibres in most instances. Motor endplates were seen which were supplied by unmyelinated motor fibres. Somatic motor innervation of the intrafusal muscle fibres was also observed. In regard to the dorsal root component of the *quadriceps* the author found that both myelinated and unmyelinated sensory fibres were seen in the nerve going to the muscle. The receptors of muscle were observed, namely, the muscle spindles, tendon spindles, Pacinian corpuscles and endings of small myelinated and unmyelinated fibres. The sympathetic component of a small muscle of the hind foot is described. Unmyelinated sympathetic fibres and endings were seen in the vascular plexuses, but no unmyelinated sympathetic fibres were seen ending hypolemmally in the small muscles of the foot. The author claims that he has shown that it is quite possible to mistake various types of normal and regenerating somatic fibres for sympathetic nerve fibres. The author gives a review of the literature on the subject and the article is accompanied by an extensive bibliography.

The Origin of Myofibrils.

S. BURT WOLBACH (*Anatomical Record*, January 25, 1928) presents a contribution to the literature upon the structure and histogenesis of striated muscle, based on the study of two tumours of striated muscle, one a congenital rhabdomyoma of the heart and the other a malignant tumour of skeletal muscle. In both there were present tumour cells in process of mitosis and cells exhibiting all of the structural details of adult striated muscle and therefore all of the intermediate stages in the developmental sequence. With regard to the origin of myofibrils he states that in brief and excluding theories, one group of investigators sees myofibrils first in the form of homogeneous fibrils, another group sees a preliminary granule or discontinuous filamentous stage in linear arrangement followed by fusion into fibrils. The prevailing opinion among the second group is that the antecedent granules and filaments are of mitachondrial origin. In this present paper the author puts forward the opinion, supported by the size, morphology and staining properties of the structures under consideration, that the granules which give rise to myofibrils, are of central apparatus or centriole origin. He says that the idea that the myofibril may be of central apparatus derivation is startling, in view of the fact that such an origin has not been suggested by studies upon embryological material. The rôle of the central apparatus in all kinetics, however, is, he states, a familiar one to cytologists.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION in conjunction with the Section of Medicine and the Section of Neurology and Psychiatry was held in the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on May 31, 1928, DR. J. E. V. BARLING, the President, in the chair.

Encephalitis.

DR. A. J. COLLINS read a paper entitled: "Epidemic Encephalitis" (see page 72).

PROFESSOR W. S. DAWSON read a paper entitled: "The Mental Aspects of Encephalitis" (see page 74).

DR. J. E. V. BARLING in inviting discussion on the subject of the two papers, said that with the possible exception of syphilis, encephalitis was associated with a greater number of symptoms than any other disease of the nervous system. In many patients who manifested obscure nervous phenomena encephalitis equally with syphilis must be considered as a possible cause. He asked the speakers whether they had noted the high sugar content of the cerebro-spinal fluid in acute encephalitis. It was stated by many authorities that this was common, whilst others attached less importance to this observation. If at all common, it would be a fact of considerable diagnostic significance.

DR. OLIVER LATHAM demonstrated a slide of a section of the brain of a woman who had been admitted to hospital with the diagnosis of septic abortion. She had died of a condition like eclampsia. The brain and the spinal cord had been examined after death and it had been found that she had died of encephalitis. The first thing to which he wished to draw attention, was the extraordinary injury to and destruction of the neurones. At first sight these changes might have been regarded as *post mortem* changes, but the excellent preservation of the delicate early neuroglial reaction negatived this. Besides the cortical cells were not so affected as the cells near the aqueduct of Sylvius where the neuroglial reaction also was more pronounced. Here, too, were many mononuclear cells as well as a fine gliosis, all constituting the earlier changes in encephalitis.

In addition there were great changes in the blood vessels. The blood vessels even at this early stage showed widespread hyaline degeneration associated with perivascular hæmorrhages and obliteration of the lumens and œdema. He had not often in Australian experience been able to recognize degeneration of the pigmented cells of the *substantia nigra*, but in this case it was very obvious with characteristic scattering of the pigment granules. There was also widespread neuronic damage in the central nuclei.

Dr. Latham spoke of the possibility of cure. It had been pointed out that cures in general paralysis had been recognized after attacks of malaria and typhoid fever. In typhoid fever and malaria there was a fundamental reaction and proliferation of the reticulo-endothelial system exemplified in the brain by the proliferation of the endothelial cells of the smaller blood vessels. The apparent peeling off of these cells into the lumen of the vessels *et cetera* had been described as an actual migration to devour the parasites concerned. Recent American work suggested that this was part of the mechanism whereby malaria favourably influenced general paralysis. It certainly mechanically allowed more fluid to permeate the denuded vessel wall and let antibodies reach commonly inaccessible regions of cortex. Such phenomena might explain the common presence of perivascular hæmorrhages and œdema in the encephalites. As malaria did not benefit *encephalitis lethargica*, it was possible that here the antibodies were inadequate or the causal organism resisted the action of the endothelial cells.

Before concluding Dr. Latham again referred to the changes in the blood vessels. Prior to his acquaintance with *encephalitis lethargica* and kindred experimental work on animals, the extreme hyaline and calcarious degeneration, found in the blood vessels in these diseases so early as the first week or ten days, seemed incredible. Vascular phenomena dominated the earlier stages and the mechanical effects of the œdema and perivascular hæmorrhages might account for many of the clinical phenomena apart from specific toxic action on the neurones. Lastly gross cerebral and especially intraventricular hæmorrhages were often met with, while as yet the neuronic and glial manifestations had scarcely begun.

PROFESSOR A. E. MILLS stated that unless they had a clear conception of the pathology of the disease they could not understand its symptoms. Dr. Latham had made reference to the early vascular changes which occurred in epidemic encephalitis and he (Professor Mills) would like to emphasize the importance of the vascular changes which occurred in the disease. To him it appeared that two factors had to be taken into consideration, vascular disturbance and toxæmia. Toxins, circulating in the blood, might cause death of the cells or vascular changes as a result of the disease might be so profound that little blood would reach the tissues, the cells of which would suffer from starvation and would degenerate and die. The so-called cuffing of the vessels by accumulation of lymphocytes in the perivascular spaces would tend to bring about a narrowing of the lumen of the vessels and so lead to deficient blood supply and consequent starvation of the tissues. So that whether it were the effect of toxæmia or the effect of blood deficiency the result would be the same, namely, degeneration and death of the tissues.

If toxæmia were held to be the only cause of the symptoms, then in those cases in which symptoms appeared long after the infection, it must be acting continuously but very slowly on the cells and so cause their death or else it must be the result of the action of the infective agent on the blood vessels causing those inflammatory changes which were characteristic of the disease. The symptoms of the disease resulting from these effects would depend upon the portion of the brain affected. If it were in the occipital lobe, vision might easily be disturbed; if in the mid-brain paralysis of the ocular muscles might take place; if it were in the basal ganglia, those symptoms characteristic of the Parkinsonian syndrome would appear.

Professor Mills referred to the condition known as *encephalitis periaxialis diffusa*. They were extraordinary cases. The changes were usually very widespread. At times the symptoms of stupor and lethargy were unaccompanied by other symptoms. For a time the children assumed an altered character. He referred to a little boy who became cunning and stole things. He had given a grave prognosis and had expected that the moral delinquency would be permanent. Later, however, the child had recovered and there was no trace of this altered nature.

He did not agree with Dr. Collins's remarks concerning the limited use of lumbar puncture. He had seen many cases in which the benefit derived from lumbar puncture was very pronounced. It was a curious fact, too, that in many cases of encephalitis there was a great excess of cerebro-spinal fluid and the characteristic symptom in such cases was lethargy. In other cases in which there was no excess of cerebro-spinal fluid, lethargy might be present, but it was then due to a diffuse inflammatory condition of the cortical grey matter.

Another fact difficult to understand was that though syphilis, like epidemic encephalitis, had a profound effect upon the vessels and by its poison produced degeneration of nerve cells and fibres, yet affections of the basal ganglia, giving rise to the Parkinsonian syndrome, were very rarely met with in cerebral syphilis.

DR. H. C. ADAMS called attention to the frequency of epidemic encephalitis in England and in America. It was increasing in Australia and demanded their careful study. He wished to refer to a matter of treatment. He had seen many patients at the Sydney Hospital suffering from encephalitis. He had found that as long as there was increase of pressure of the cerebro-spinal fluid, lumbar puncture always afforded relief. In this he differed from Dr. Collins. He spoke of a boy who had been an inmate of a suburban hospital and had been seen by a psychiatrist. The diagnoses made of his condition had been epilepsy and

cerebral tumour. When the boy was admitted to the Sydney Hospital, there were convulsive movements of one arm and one leg. Lethargy was present. Lumbar puncture was performed. There was increased pressure, but no increase in cells. The lumbar puncture had been the turning point of his illness. He had begun to improve at once and after a relatively short time he had been discharged well. A woman had been admitted to hospital said to be suffering from debility from overlactation. Unilateral Parkinsonism had been detected. The treatment had consisted in measures to improve her general health and the extraction of some teeth. The latter had been undertaken on account of chronic sepsis (granulomatous infiltration) at the apices of the teeth. Great improvement had followed the extraction. Dr. Adams held that it was of prime importance to clear up toxæmias in these patients. He mentioned the views of Price as expressed in his book on oral sepsis. Unfortunately many patients were admitted to hospitals without having their condition diagnosed. The result was depressing in adults and disastrous in children. In conclusion Dr. Adams raised the question of the source of infection in some of the patients. Many of the patients he had seen had come from the backblocks. He remembered a boundary rider who had been brought in with encephalitis. This man had lived an isolated life. He had died and the diagnosis had been confirmed at autopsy. He thought that the diagnosis in the early stages was difficult.

DR. G. A. HARDWICKE spoke of 135 patients who had been treated for epidemic encephalitis at the London Hospital between 1919 and 1921. The mortality was 26%. In 95% of the patients one or more of the sequelæ had occurred. The mortality from the sequelæ varied between 15% and 20%. He gave further information concerning the type of sequelæ encountered in these patients; 25% of the sequelæ were of the nature of symptomatic *paralysis agitans*. In regard to treatment they had found that twenty cubic centimetres of a 4% solution of sodium salicylate given intravenously was of value. At times they had injected from fifty to one hundred cubic centimetres of a 10% to 15% solution of sodium chloride when the lethargy was well developed. Professor Mills had spoken of a patient who had become a thief during the later stages of the illness. He, too, had seen a boy, aged ten years, who had become a thief. Like Professor Mills's patient, this boy had regained his former conduct when he recovered from the illness. None of the previous speakers had mentioned the gold sol test in encephalitis. This test had been applied, but no characteristic curve had been discovered. The best treatment for the salivation commonly associated with Parkinsonism was pilocarpine.

DR. J. INGLIS ROBERTSON wished to know what was to be done with those children who became morally deteriorated as a result of epidemic encephalitis. They often became potential criminals. It would be unjust and irrational to punish these children for offences. Something should be done to protect them. A system of notification might be adopted, in order that the police would have information concerning the past history of the children and their irresponsibility.

DR. E. L. SUSMAN had been much interested in Dr. Collins's reference to the smouldering stage of encephalitis. This form presented great difficulties in diagnosis. He wished that Professor Dawson had dealt in greater detail with the differential diagnosis. At times the signs were quite atypical, closely resembling the symptomatology of neurasthenia. There might be industrial inefficiency, slowness and lessening power. In these patients rigidity was often of the utmost importance. Sometimes this rigidity was confined to the muscles concerned in the production of speech, producing a characteristic alteration, a monotonous tone. Another important sign was the change in some of the eye muscles. When the patient was asked to follow the finger with the eyes, it was found that instead of a steady sweep across the field, there was a *staccato*, jerky movement, "by numbers" as it were. A valuable pupillary sign was the absence of reaction to convergence. A previous speaker had mentioned Schilder's disease, by way of comparison. He thought that Schilder's disease, *encephalitis periaxialis diffusa*, was a chronic primary nervous degeneration, while *encephalitis lethargica* was an acute hæmorrhagic inflammation of the brain pathologically distinct. Such comparisons tended only to increase the difficulties of an already difficult subject. He held the opinion that lumbar puncture was the only treatment that gave satisfactory results and then only if performed frequently.

DR. R. A. NOBLE spoke of the parallelism between encephalitis and syphilis. The spirochæte attacked the vascular system, while the virus of encephalitis attacked the chorioid plexus and the ependyma. He was convinced that the main damage was done at the time of the attack. There was no definite evidence that the virus persisted. The most important matter was early diagnosis and the application of lumbar puncture. The general practitioner had to make the diagnosis. By the time the patient reached the specialist the diagnosis was easy. The prognosis was not bad if the diagnosis was made at the early stage. It was dangerous to look upon every patient with irregular nervous symptoms as hysterical. At times the condition proved to be encephalitis. One patient had been troubled with upward rotation of the eye. It seemed as if this man had good reason to adopt an hysterical trick. Later it had been found that the patient had developed Parkinsonism. Unilateral Parkinsonism was not uncommon, but nearly always progressed to bilateral changes. In children inversion of the sleep rhythm was common. Deterioration of conduct also occurred frequently.

DR. A. S. WALKER spoke of the difficulty in obtaining reliable histories at times of the acute illness of encephalitis. The patient with lethargy often failed to remember any acute illness and sometimes the relatives could give no help. If the relatives were closely questioned it was often possible to elicit a history of so-called influenza with nocturnal restlessness as an outstanding feature. In larval cases there might be slight ophthalmoplegia. He recalled the details of the illness of one man who had had epidemic hiccup with ophthalmoplegia and diplopia. Later there was a distinct moral change. He asked whether Dr. Collins could give any information concerning the ultimate fate of persons who had had epidemic hiccup. In conclusion Dr. Walker said that he had seen two patients with Parkinsonism who had been subjected to the operation of ramisection. Neither had derived any benefit from the operation.

DR. COTTER HARVEY wished to have an expression of opinion concerning the superimposition of functional symptoms on the Parkinsonian condition. He had seen unilateral paresis in a patient with Parkinsonism, with inability to walk. Under appropriate suggestion the patient had been induced to walk quite well. He thought that the improvement after ramisection in this condition might be ascribed to the suggestive effect of the operation. It had been performed on one patient of his and the operation on one side only had been followed by definite improvement which, the patient had asserted, was equal on both sides. After two months, however, the patient relapsed. It seemed as though the improvement had been a functional one.

DR. C. A. HOGG said that in the mental hospitals and at Broughton Hall they saw what might be called residuals. The general practitioner saw patients with early epidemic encephalitis and the psychiatrists saw mostly those who ultimately had mental and nervous sequelæ. The only hope of the patient, therefore, was in the hands of the general practitioner who should endeavour by early diagnosis to prevent the development of sequelæ.

Dr. Collins replied to several of the speakers. He had had no experience of an increase of glucose in the cerebro-spinal fluid. He regarded Professor Mills's hypothesis of a basic causation in the vascular system as most attractive. He (Dr. Collins) had always held that the nervous elements shared in the same toxæmia as the cerebral vessels. In regard to the criticism concerning the value of lumbar puncture, he had, perhaps, not made his meaning clear. He had intended to convey the impression that lumbar puncture did good only when there was cerebro-spinal irritation. He had been very interested to learn of the experience of other practitioners. He had not learned of any sequelæ of epidemic hiccup. He trusted that there were none. Epidemic hiccup was probably an acute infec-

tion of that part of the spinal cord from which the phrenic nerves arose, namely, the third, fourth and fifth cervical segments.

Professor Dawson regretted that very little had been achieved towards finding a solution to the medico-legal problem of the morally defective child. In England, in order chiefly to meet the patients with post-encephalitic changes in character they had passed an amendment to the *Mental Deficiency Act* to provide for moral defects caused by injury or disease up to the age of eighteen years. He held that every case would have to be argued out and dealt with in the court on its merits. In regard to the functional elements encountered in patients suffering from encephalitis and its sequelæ, he pointed out that neurasthenic symptoms were very common and had been referred to by him in regard to the differential diagnosis. These symptoms often included the occurrence of abnormal fatigue and were frequently the precursors of the Parkinsonian syndrome. It was necessary to weigh all the factors after prolonged observation. In this way alone could an accurate diagnosis be achieved in many cases. Head had pointed out that when the bodily tone was lowered through infection, exhaustion or emotional stress, vigilance, as he termed it, was impaired and function became less efficient. Disability from organic disease was often found to vary according to the general condition of the patient and his state of well-being. If the general tone were good he could make an effort and the disability would be lessened. At other times tone might be lowered and symptoms were more severe and they were inclined to speak of a "functional" element in the case. It was extremely difficult to distinguish between the physical and the psychological signs and symptoms and this difficulty added to the problems of the physician in arriving at a diagnosis.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on March 2, 1928, DR. EUSTACE RUSSELL, the President in the chair.

Physiology of the Biliary Tract.

DR. H. S. MCLELLAN read a paper entitled: "The Biliary Tract: Recent Advances in Knowledge" (see page 80).

Transposition of Viscera.

DR. L. M. MCKILLOP showed a woman, aged sixty years, who had had nine confinements. She had consulted Dr. McKillop in August, 1927, for recurrent attacks of burning pain in the hypogastrium. A provisional diagnosis of colitis, due to appendicitis, had been made. Upon opening the abdomen in the mid-line Dr. McKillop had found that the appendix was inflamed, but was situated in the left iliac fossa. In this position he had also found the terminal portion of the ilium and the caecum, whilst the descending colon which bore all the evidence of a chronic inflammatory condition, was lying in the right iliac fossa. The large lobe of the liver and the gall bladder had occupied the left hypochondrium and a subsequent X ray examination had revealed transposition of all the thoracic viscera.

Dr. McKillop also showed a woman, aged thirty-two years, upon whom he had recently operated for a large fibroid tumour of the uterus. In this patient he had found a state of affairs exactly similar to that described in connexion with the previous patient.

Arterio-Venous Aneurysm.

The next patient shown by Dr. McKillop was a railway ganger who had been struck fourteen years previously by a splinter of steel immediately above and to the inner side of the antecubital fossa of the right forearm. Shortly after this injury a pulsating swelling had formed on the inner and anterior aspect of the upper part of the arm and had persisted with little or no alteration and with certaintly no inconvenience to the patient. The patient had come for operation for prolapse of the hepatic flexure and of the ascending colon. The aneurysm between the brachial artery and the median basilic vein had been found during physical examination.

Osteosarcoma of the Radius.

Dr. McKillop also showed a specimen of an osteosarcoma which had been removed from a boy of thirteen years. This lad had been treated in October, 1927, for a simple fracture of the left radius by Dr. P. N. Macgregor. The fracture had been put up and X ray examination had shown that it was in an excellent position. In December, 1927, a painless swelling had appeared in the region of the wrist. This had gradually increased in size. An X ray examination had been made by Dr. A. J. Reye who found what had appeared at first sight to be an osteomyelitis in the old line of the fractures. In view, however, of the absence of pain and fever, the condition had been regarded with suspicion and a further X ray examination had been made at the end of January. Close examination of one film had revealed the presence of bony spicules lying at right angles to the long axis of the radial shaft; the inner aspect of the radius had appeared to be completely eroded. After making a diagnosis of sarcoma, Dr. McKillop had amputated the arm below the insertion of the deltoid muscle. Dr. J. V. Duhig had examined the specimen and had found a sarcoma which exhibited osteogenetic changes.

Spontaneous Rupture of the Rectum.

Dr. McKillop also showed a specimen of a rectum which had undergone spontaneous rupture. This specimen aroused great interest amongst those present. Dr. McKillop said that it was probably the only existing specimen of such a nature and that no reference to such a condition could be found in the available literature. He said that he hoped to report the case in full at a later date.

Astragalectomy.

Dr. McKillop showed a boy, aged fifteen years, from whom he had removed the astragalus eighteen months previously on account of a badly comminuted fracture. The patient had a good range of movement and the functional result was excellent.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Nairn, William Archibald Jock, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.
Homewood, Arthur Norman, M.B., Ch.B., 1915 (Univ. Edinburgh), Millicent.
Lendon, Alan Harding, M.B., B.S., 1927 (Univ. Adelaide), Adelaide.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Abdullah, Anthony Dominic, M.B., Ch.M., 1926 (Univ. Sydney), 267, Oxford Street, Darlinghurst.
Ginsberg, Maurice William, M.B., B.S., 1927 (Univ. Sydney), Sydney Hospital, Sydney.
George, Noel Francis, M.B., B.S., 1928 (Univ. Sydney), Koorawatha.

Obituary.

JOHN TIMOTHY KENNEDY.

THE late Dr. John Timothy Kennedy, of Cobram, who died after a short illness on February 19, 1928, was born at Listowel, Kerry, Ireland, and received his medical education at the Edinburgh University and the Rotunda Hospital, Dublin. Graduating in 1888, he soon set sail for Australia and on arriving in Victoria, commenced practice in Cobram, on the Murray. This was the centre of a large district extending many miles into New South

Wales and the young Irish doctor soon established himself in the regard of his people and to the day of his death was held in the highest esteem by the whole community. He leaves a family of eight, his wife having died some years ago. Of the sons, three are in the medical profession, Dr. J. A. Kennedy, of Shepparton, and Dr. Gerald and Dr. Adrian Kennedy, of Cobram.

JOHN CAM WIGHT.

DR. JOHN CAM WIGHT died on June 2, 1928, at Kyabram after some considerable period of ill health during which he endeavoured to carry on his practice. He was the son of Mr. E. Byam Wight, of Melbourne, a very old colonist. Graduating at the Melbourne University in 1884, he continued his studies at Vienna and specialized in laryngeal work. Returning to Melbourne in 1888, he started practice in Balaclava, but finding the climate unsuitable to his health (he had been showing a tendency to asthma), he removed to Kyabram in 1893, then but a small village. He soon commanded the practice of a large district which ere long became one of the most thriving in Victoria. Dr. Wight was to a great extent responsible for the advancement of the town and furthered its interests in every way. At the time of his death he was the President of the Goulburn Valley Subdivision of the Victorian Branch of the British Medical Association and presided at the May meeting of the Subdivision.

Dr. J. W. Florance writes:

The passing of these two beloved physicians was felt, of course, more immediately in their respective towns and districts. Here they had established themselves shortly after their graduation and here they lived and worked for the good of their communities. So they were respected and trusted and their names ever stood for honour and sincerity.

But to me, their colleague, there has come a void incapable of being filled. All these past forty years we had felt we could call on one another in our troubles. We worked together in the old days when roads were almost impassable and operations had to be performed on hastily improvised tables, also in recent years when private hospitals and trained nurses have made things easier. At every meeting discussion led to enjoyment in one another's company and the strengthening of the bond of friendship. The formation of the Goulburn Valley Medical Society, later to become a Subdivision of the Victorian Branch of the British Medical Association, was brought about chiefly by these meetings.

As practitioners their work was always practical and followed the most recent teachings, for they never allowed themselves to become fossilized. As members of the British Medical Association they took the keenest interest in its welfare and were to a great extent responsible for the strong membership existing in the Goulburn Valley in early days.

Correspondence.

VENEREAL INFECTION.

SIR: It is extremely gratifying to see that some members of our profession are at last displaying an interest in the serious problem of the increasing prevalence of venereal infection.

Cancer and tuberculosis are subjects of endowed research and everyday discussion, but neither are responsible for so much havoc as venereal diseases. Sir James Barrett in his foreword to my book on this subject gives the following figures relating to mortality in England and Wales during the year 1924: Total deaths, 473,235; deaths due to syphilis, 60,335; deaths due to cancer, 50,389; deaths due to tuberculosis, 41,103.

Statistics, however, should not be relied upon for forming conclusions regarding venereal diseases, more particularly gonorrhœa. Experience in dealing with large numbers of cases is surely the more reliable guide. In this connexion the results of the study of some five hundred delinquent children as quoted in Dr. McArthur Brown's letter (THE MEDICAL JOURNAL OF AUSTRALIA, June 30, 1928) gives us much food for thought. That nearly 50% of these adolescents should be found to be suffering from venereal disease is astounding. Undoubtedly many of the cases are victims of environment, but others are surely the result of ignorance, ignorance of the risks of illicit intercourse and ignorance of methods of personal prophylaxis.

There is a large field for trained social workers amongst adolescents and the richest harvest in my opinion is to be reaped at the onset of puberty by education and moral influence along the lines of proper control of the sexual appetite. Unless the minds of youth are moulded at this stage, it often happens that later no religious, ethical, idealistic, educational or social influences will prevail against the indulgence of their acquired or inherited passions.

The significance of physiological phenomena such as nocturnal emissions, menstruation *et cetera* should be clearly explained to all adolescents and, moreover, they should receive instruction in methods of preventing and means of recognizing the infections likely to be acquired after any act of illicit intercourse. The teaching of sexual physiology I regard as unimportant and likely to be of little avail in preventing these diseases. The physiology and mechanism of reproduction, except in mere outline, should always remain in my opinion shrouded in mystery. In the education of the young it is not desirable to emphasize the sordid details and gruesome complications of these diseases, but it is most essential that everyone should be able to recognize the infection in its earliest stages and be acquainted with methods of protecting themselves from disease.

Young men and women are kept in ignorance of these important matters merely on account of prudery and false shame on the part of those best able to advise them. It is foolish to preserve one's plaster saints at the expense of youth's physical wreckage!

Another point seems worthy of comment. Notification is compulsory in New South Wales and this measure I am sure is achieving a contrary purpose to that for which it was intended. The fear of publicity leads to concealment and many patients with gonorrhœa prefer to treat themselves rather than consult a doctor. After the acute symptoms have subsided, their former sexual life is resumed (gonorrhœa is unfortunately a sexually exciting disease). They thereby infect others and become a menace to society.

Chemists in this State are prohibited, moreover, from supplying any drug or application for the prevention of venereal disease, except on the prescription of a medical practitioner. Surely this is a retrogression!

Prophylactic facilities are available day and night from most specialists in all large cities, but they are not often availed of. It is not generally known that disinfection of the parts (including the *fossa navicularis*) after exposure with a cream containing calomel, thymol and mercuric oxycyanide will prevent both gonorrhœa and syphilis in 99% of cases. These packets should be available at any chemist and instead of prohibiting their sale as at present, governmental authority should exercise supervision in their manufacture so that the public may be assured of their efficacy and potency. I do not think that promiscuous sexual relations would be any more common than they are at the present time, nor would the use of these packets be construed as "licences for pleasure."

Another important reform urgently desired is the introduction of a uniform standard of cure for gonorrhœa. Many patients are being discharged as cured by their medical attendant while still in an infectious stage.

Two tests I regard as essential in every case are: (i) The prostatic pus test, (ii) semen culture. Urethroscopic and complement fixation examinations are important but subsidiary.

Vast changes in methods of diagnosis and treatment in venereal diseases have been introduced in recent years and these should be familiar to every practitioner who undertakes their treatment.

Yours, etc.,

ROBERT V. STORER.

231, Macquarie Street, Sydney.
July 1, 1928.

Corrigendum.

DR. LUCY M. BEYCE has pointed out that a mistake was made in the references to her article: "A Note on the Varieties of Vaginal Bacilli Met With in an Investigation of the Flora of the Female Genital Passages," which appeared in the issue of June 30, 1928. The second reference should be as follows: E. Löwi: *"Ueber den Bacillus crassus Lipschütz," Wiener Klinische Wochenschrift*, August, 1920, *Band* XXXIII, *Seite* 730.

Diary for the Month.

JULY 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 25.—Victorian Branch, B.M.A.: Council.
JULY 26.—New South Wales Branch, B.M.A.: Branch.
JULY 26.—South Australian Branch, B.M.A.: Branch.
JULY 27.—Queensland Branch, B.M.A.: Council.
AUG. 1.—Victorian Branch, B.M.A.: Branch.
AUG. 1.—Western Australian Branch, B.M.A.: Council.
AUG. 2.—South Australian Branch, B.M.A.: Council.
AUG. 3.—Queensland Branch, B.M.A.: Branch.
AUG. 7.—Tasmanian Branch, B.M.A.: Council.
AUG. 9.—Victorian Branch, B.M.A.: Council.
AUG. 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 10.—Central Southern Medical Association, New South Wales.
AUG. 10.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. Michael Leonard Devaney McKeon (B.M.A.) has been appointed Acting Government Medical Officer at Bundaberg and an Acting Health Officer under *The Health Acts* 1900 *to* 1922, of Queensland.

* * * *

Dr. Eugene Abraham Matison (B.M.A.) has been appointed Honorary Surgeon for Diseases of Ear, Nose and Throat at the Parkside Mental Hospital, South Australia.

* * * *

Dr. Owen Meredith Moulden (B.M.A.) has been appointed Honorary Gynæcologist at the Parkside Mental Hospital, South Australia.

* * * *

Dr. Frederick Steele Scott (B.M.A.) has been appointed Honorary Dermatologist at the Parkside Mental Hospital, South Australia.

* * * *

Dr. F. W. Cotton (B.M.A.) has been appointed Quarantine Officer at Wyndham, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

DEPARTMENT OF PUBLIC HEALTH, VICTORIA: Clinical Tuberculosis Officer.
MENTAL DISEASES HOSPITAL, NEW NORFOLK, TASMANIA: Junior Medical Officer.
ROCKHAMPTON HOSPITALS BOARD: General Medical Superintendent.
UNIVERSITY OF HONG KONG: Reader in Biology.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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